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COMMUNICATIONS

A PEDIGREE OF MICROPHTHALMIA WITH MYOPIA AND CORECTOPIA

BY
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ABERDEEN.

NOT very many pedigrees of microphthalmia have been published, and a number of these refer only to cases in a single sibship or else to a parent and child. In the list of literature appended to this paper, there are five pedigrees, however, that show continuous descent in three generations (Page, Mayerhausen, Martin, Schaumberg, Ravá), one pedigree that shows discontinuous descent with affected members in three generations (Barkan), and another both continuous and discontinuous inheritance with affected members in three generations (Cunier), but it does not appear from the descriptions of the cases that any of the microphthalmic individuals in these, or any of the pedigrees were myopic. Furthermore, the inheritance of microphthalmia with corectopia is apparently uncommon. Harman recorded a pedigree where a man, with two of his daughters, and his half sister had microphthalmic eyes and corectopia, but this is the only instance known to me. The refraction of the eyes in his cases is not mentioned. Krükow recorded the cases of two microphthalmic brothers, one of whom had corectopia in his left eye. Ischreyt mentioned microphthalmia in a mother and daughter. The position of the pupils of the

former were eccentric upwards and inwards, that of the latter was not stated. von Ammon*, in 1841, published the cases of two microphthalmic brothers, one of whom had eccentric pupils upwards and inwards and the other centrally placed pupils. He also described and figured several isolated cases of microphthalmia with corectopia. Best,[11] more than fifty years later, in referring to von Ammon's view that the association of microphthalmia and corectopia was exceedingly frequent, stated that later authors almost never observed it, but, on the other hand, that a characteristic form of corectopia was associated with ectopia of the lens and tremulous iris. Such cases are often myopic and constitute Best's third group. His second group consists of corectopia with some common developmental anomalies of the eye, such as buphthalmia and microphthalmia.

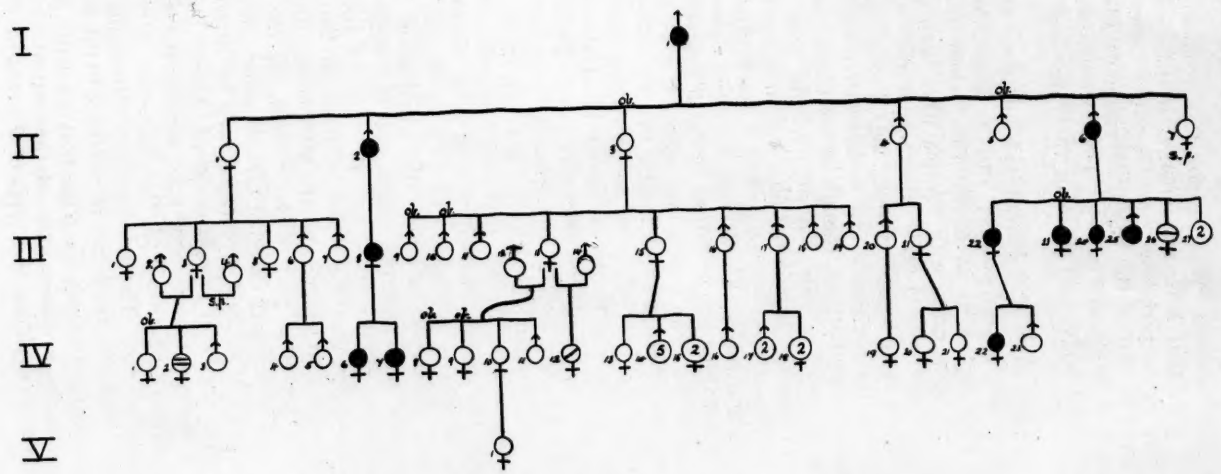
The pedigree which forms the subject of this paper shows direct descent of microphthalmia with corectopia and myopia from a male proved to have had myopia and reported by two of his sons and a grand-daughter, all of whom were microphthalmic, to have had small eyes like their own. All of these abnormalities were present in each affected individual and appear in probably four consecutive generations, and certainly in three. The microphthalmia and corectopia invariably affected both eyes, and the pupils were markedly displaced upwards and inwards in each case, excepting possibly one in which they were described as being "eccentric towards nasal side." From a perusal of their notes, it is evident that the microphthalmic cases present several of the characters that occur in Best's third group of corectopia, but, the presence of microphthalmia would assign them a place rather in his second group.

There was no coloboma of lens, iris, choroid, optic nerve, or eyelid in any case, but the presence of an eccentric pupil upwards and inwards was invariable, and in some cases the anterior surface of the iris presented peculiarities. In one case (III. 22) the circulus iridis minor was absent at the inner upper part in the right eye, and at the outer part in the left eye. In another (III. 24) the circulus iridis minor was complete in the left eye, but absent at the nasal side in the right eye. A mother (III. 8) and her two daughters (IV. 6 and 7), had irides of unusual appearance. The peripheral zone, smaller than the pupillary zone, was of a darker grey colour than the pale grey of the larger pupillary zone which ended in a ragged edge where it met the darker area.

The measurements of the transverse diameters of the corneae were from eight to ten millimetres. The lens was generally clear, though there were some exceptions. In a man, aged 68, there were

* *Vide* Gescheit.[4]

MICROPHTHALMIA



NO CONSANGUINITY

⊜ DEAF-MUTE
⊖ INSANITY
⊘ EPICANTHUS

cortical striae and posterior polar cataract, a woman, aged 41, had an opaque dislocated lens, and another woman a secondary cataract; in two cases peripheral dot opacities were present. Glaucoma occurred in two of the cases. Nystagmus was noted in five of the cases, and its absence in two. There were no deformities of other parts of the body, and none of the cases was mentally affected.

The myopia in these cases is of considerable interest. Most cases of microphthalmia are hypermetropic, and in some the hypermetropia is of high degree, as in Roll's[1] case, where there were twenty dioptres, yet some cases with myopia have been recorded, such as Brailey's[2] case with twelve dioptres in the right eye, Nieden's[3] case which was highly myopic, and Pyle's[4] case with twenty dioptres. In the pedigree under consideration myopia was present in all the microphthalmics, though in two cases, one a child of three years of age, one eye only was myopic, and from a study of the literature of microphthalmia the question arose whether the myopia was of the usual form—axial, or had it some other origin situated in the lens or cornea? Becker[5] attributed the presence of myopia in a microphthalmic boy of fifteen years, to the very short radius of curvature of the small cornea and regarded it as a curvature myopia. In some of the microphthalmic cases in the pedigree under consideration the cornea appeared to be flatter than usual, yet in two cases where the radius of curvature of the anterior surface was measured by the ophthalmometer it was found to be rather less than the 7.8 mm. and 7.7 mm. given by Donders[6] as the average in man and woman respectively, but not sufficient to account for the degrees of myopia that were present. Mayou[7] examined an enucleated microphthalmic eye of a six months old child and found a lenticonus posterior. In view of Mayou's case the possibility was considered whether the explanation of the myopia in the pedigree now reported might not be found in an hereditary malformation of the lens, but in none of the cases examined for this condition was there any evidence of lenticonus posterior. There is reason to believe that the myopia was produced by elongation of the posterior part of the eyeball, for the usual fundus changes found in ordinary cases of myopia were present in several of the cases, and in at least two cases, seen at intervals, a marked increase in the degree of myopia occurred. Querenghi[8] saw in the fundus of a microphthalmic eye that had six dioptres of hypermetropia, appearances that had all the characters of a progressive posterior staphyloma. The association of corectopia with microphthalmia is in a sense contradictory in reference to the state of the refraction, for in microphthalmia the refraction is usually hypermetropic, whilst in corectopia with ectopia of the lens it is often myopic, and there is some evidence of ectopia of the lens in several

of the cases in this pedigree. Iridodonesis was present in four of the cases, the edge of the lens was visible in the pupil of one of them, an opaque lens showed partial dislocation in another, and the anterior chambers were each of unequal depth in a fifth case (III, 24). In all of the cases vision was poor even after correction of the error of refraction. The lowest degree of myopia present was 1 D., found in a girl, seven years of age, but it had increased to 7 D., by the time she was fourteen. The highest degree of myopia was 20 D. The combination of albinism* and microphthalmia, as seen in the child IV. 7, has been recorded by Posey[9] in a four year old female child. Of other members of the pedigree without microphthalmia II. 1, and her son III. 7, were highly myopic. IV. 2, is a deaf-mute, and III. 26, one of a microphthalmic sibship, was mentally affected and died in a lunatic asylum.

The following is an account of the individual cases of microphthalmos and of some other cases in the pedigree:—

I 1. G. W.——, male, had small eyes. This is vouched for by his son II 2, who said he was sure that his father had small eyes, and also by his son II 6, and by his grand-daughter III 8. In a register book at the Aberdeen Royal Infirmary his name was entered on June 23, 1890, and his age given as 68. In the column for diagnosis, instead of the name of some disease as in other cases, was inserted "myopia, choroidal atrophy, tremulous iris, striae and posterior polar cataract, large staphyloma posticum." The cornea was not mentioned. He was illegitimate. In 1897 one of his sons reported that he (I 1) had been blind for five years.

II 2. G. W.——, male, age 49 (1895), always near sighted, complained that the vision of his right eye had been failing for one week, and that everything appeared green to that eye. The squint of his right eye—internal concomitant—was of long standing. Corneae are small, transverse diameters=9.5 mm. Refraction: myopia, R. more than 11 D., V. not improved beyond 3/36; L. 20 D. in vertical meridian and 25 D. in horizontal meridian, V. with correction=6/60; pupils eccentric towards nasal side, iridodonesis; posterior staphyloma with choroidal atrophy surrounding O.D. in each eye, in L. extensive choroidal changes at macula, no vitreous opacities. When seen again 1904: R.V.= hand movements, faulty projection, T+1, A.C. shallow, P. oval, sluggish to light. L. T. n; O. D. n; neither eye moves fully outwards. In 1911: R. T. + 3, very shallow A.C.; L.A.C. deep, P. contracts readily to light, upper margin of lens visible in upper part of P., lens becoming opaque, V=counts fingers at 1 foot. In

* Schultze[10] found in the larvae of the salamander albinism associated with microphthalmia, and he refers to the presence of albinism with microphthalmia in the normal proteus anguinus.

1920: no p.l. in either eye, no nystagmus, much corneal opacity, R. T. n., L. T.+2.

II. 6. J. W——, male, age 41 (1897), complained of inflammation of his right eye off and on for one year. The sight of this eye failed all of a sudden in boyhood. Corneae small, transverse diameter = 10 mm., R.V. = no p.l., cornea nebulous all over; L. myopia 20 D., V. with correction=3/60, is wearing −12 D. and −16 D. lenses reversible. Ps. ectopic far upwards and inwards, iridodonesis; no details of right fundus. L. myopic crescent; R.T.+1, L.T.n., left lens hazy.

III. 8. Mrs. A. W——, age 21 (1896), was always near sighted and had never worn glasses. Corneae small and clear, no evidence of conical cornea. Refraction: R, myopia 11 D. in vertical meridian and 13 D. in horizontal meridian, V. with correction = 6/36 not fully; L, myopia 14 D. in vertical meridian and 9 D in horizontal meridian, V. with correction=6/24 not fully; large white myopic crescent in each. Seen again in 1907, "sight getting worse." R.V. <6/60; L.V. 6/60 with correction. In 1920: R.V. with −11 D = 2/60; L.V. with −11 D = 2/60; corneal transverse diameter=9 or 9.5 mm. in each, corneal curvature: the ophthalmometer shows in R. that radius of cornea is 7.6 mm.; in L. 7.3 mm.; sclero-corneal junction is well defined; Ps, ectopic up and in, are not quite circular; no nystagmus; no iridodonesis, surface of iris pale grey with a much darker peripheral zone, a well defined irregular line where the two zones meet; lenses clear with exception of a few small dots below, no evidence of lenticonus posterior; optic discs normal with large area of choroidal atrophy surrounding them, and patches of complete and partial choroidal atrophy; some vitreous opacity; T.n. A.Cs. not very shallow; eye movements full; lacrymal apparatus normal; palpebral fissures measure 27 mm. in length. When the upper lid is raised and she looks downwards the eyeball appears to be of a fairly large size, and similarly on drawing the lower lid down when she looks upwards.

III 22. Mrs. M.——, age 41 (1921), a delicate woman, formerly had stomach trouble for some years, pain after food, sometimes vomiting, and occasionally haematemesis. Had gastro-enterostomy performed last year for dilated stomach; corneal transverse diameter in each eye is 9 mm.; the ophthalmometer shows a corneal radius of 7·5 mm. in the horizontal meridian, corneal edge is well defined though less sharply than in a normal eye; the eyeballs are smaller than normal eyes; eyelids, eyelashes, and puncta lacrymalia are of normal appearance; eyelids not sunken back into orbit, palpebral fissures measure 24 mm. in length and 6 mm. at widest part; constant rotatory nystagmus; R.V. with −12 D. sph.=2/60, but she prefers a − 20 D. sph., reads 1 J. at about 2"; L.V.= no p.l., Ps. equal; R.P. contracts to light, but not consensually;

L.P. no contraction to light, but contracts consensually; irides bluish with much yellow in stroma; in R., corona is absent above, and in L., at outer part; left iris is tremulous, not the right iris; Ps. remarkably eccentric, both are displaced upwards and inwards, right P. is black, left P. is grey owing to an opaque lens, which is partially dislocated into the vitreous; right lens, clear, shows no evidence of lenticonus posterior; oph: R. no vitreous opacities, or evidence of hyaloid artery detected; a large white area of complete choroidal atrophy surrounding O.D. extends well outwards into macula, no coloboma of choroid; L, no red reflex; T.n. in each eye. Her vision has deteriorated, for in 1907 her notes indicate that R.V. with − 16 D. sph.=6/24; L.V. =p.l., is myopic, degree not determined; corneae, transverse diameter from 8·5 to 9 mm.; fundus changes were present in L. as well as in R.

III 23. L.W., female, age 13 (1893), for three years was an inmate of an asylum for the blind, where she was admitted in 1893. On her admission form, under the late Sir James McKenzie Davidson's initials, occurs the following: "High myopia. L. secondary cataract." Her sister (III. 22) says she had small eyes like her own, and Mrs. W.——(III. 8), her cousin, indicated that she had small eyes and nystagmus. She was blind for some years before she died, unmarried, aged 31.

III. 24. Mrs. McP——, age 21 (1903), complained of frontal headache, which was worse when reading. Corneal transverse diameters=10 mm.; L. corneal nebula. Refraction: R.Hm. 5 D. in vertical meridian and 5.75 D. in opposite meridian, V. with correction=6/12 not fully; L. My. 7 D., V. with correction=3/60; Ps, nearly circular, contract readily to light, ectopic up and in, nasal margin of R.P. is little more than 1 mm. from corneal margin; no iridodonesis; rotatory nystagmus; surface of iris in R. has an incomplete corona at nasal side; there the iris is slate colour and looks thinned, in L. the corona is complete; lenses have peripheral dot opacities; fundi normal; no coloboma of choroid or iris; T.n.; R. pupillary membrane across lower part of P. and fine pigmented dots on lens capsule. She has lupus of face. Seen again in 1907 and 1912; marked convergent concomitant strabismus of L; A.C.s deep down and out, shallow up and in; no ptosis.

III. 25. J. W.——, male, age 12 (1897), was always short sighted. Corneal transverse diameter=8 mm. in each eye. Refraction: R. myopia 7 D. in oblique meridian, and 10 D. in opposite meridian, V. with correction=2/60; L. myopia 7 D. in oblique meridian, and 10 D. in opposite meridian, V. with correction=5/60; Ps. markedly ectopic up and in, contract to light and on accommodation, approach closely to corneal margin, and are not circular; lateral nystagmus. Oph.: large crescent of complete choroidal atrophy at outer margin of each O.D., and in R. another

patch with well defined margin, at macula; in L. an opacity in front part of vitreous; ptosis, partial, about 1/3. Teeth good; arch of palate rather high.

IV. 6. A. W.——, female, age 2 (1907), holds things too near. Corneal diameter=9.5 mm. in transverse direction in each. Refraction: R. myopia 3.5 D. in horizontal meridian, and 4.5 D. in vertical meridian; L. myopia 1.50 D. in horizontal meridian, and 2.50 D. in vertical meridian; Ps. markedly ectopic upwards and inwards; rotatory nystagmus. The myopia had increased to 10 D. in each eye by the age of 7. Seen again in 1920: corneae are clear with well defined margins, curvatures of anterior surfaces look less convex than usual, giving an impression of flatness; iris surface has a dark grey peripheral zone with irregular margin where it joins a larger pale grey pupillary zone containing some yellow spots; no lens opacity, no lenticonus posterior; fundi moderately pale, posterior staphyloma of complete choroidal atrophy in each; eyeballs look small in profile, but appear to be of larger size when exposed by raising upper or depressing lower lid, when the eye is looking down and up respectively. A.C. not shallow, and is of uniform depth; no iridodonesis, slow rotatory nystagmus; lids normal; all puncta lacrymalia are present; length of palpebral fissure is 24 mm. She was born at full time; began to menstruate at age of 12.

IV. 7. E. W.——, female, age 6 (1912), corneae small. Refraction: given as, R. My. 1 D. in vertical meridan and Hm. 1 D. in horizontal meridian; L. My. 1.5 D. in vertical meridian and emm. in horizontal meridian; L. diverges; rotatory nystagmus; fundi typically albinotic and hair is very fair; she is a fairly complete albino. Seen again in 1920, myopia had increased in R. to 7 D. in horizontal meridian and 8 D. in vertical meridian, V. with correction=6/36 and in L. to 7 D. in horizontal meridian and 8 D in vertical meridian, V. with correction=6/36; corneal transverse diameters=10 mm., corneae are clear, edges well defined and curvature less marked than normally; Ps. markedly ectopic upwards and inwards, not quite circular, lenses clear, no lenticonus posterior, no iridodonesis, iris bluish grey with darker peripheral zone which has a sharply defined irregular edge where it joins the larger and much paler pupillary zone: A.C.s of moderate and even depth; posterior staphyloma in each, not noted eight years ago; eyeballs generally give an impression of smallness; constant fine rotatory nystagmus. Mother says she was prematurely born, but adds that her doctor said she was born at full time; menstruation has not yet commenced.

IV. 22. I. M——, female, age 3 (1921), born at full time; corneae small and transparent, edges well defined, no conicity, indeed they have rather a flattened appearance, transverse diameter

of each measures 9 mm., whole eyeball is too small; no nystagmus; palpebral fissure quite wide, eyelids with puncta and eyelashes have a normal appearance; irides blue, with some yellow in stroma; no iridodonesis; lenses clear, no lenticonus posterior; A.C.s of equal and usual depth; oph.: optic discs normal, fundi pale. Refraction: in R. shadows are hypermetropic, in L. myopic; holds objects close to her; Ps, black, contract to light, are markedly ectopic upwards and inwards, and are ovoid.

The next two cases, with high myopia, were examined without the knowledge that they were members of this pedigree. As there is no mention in their notes of the size of the cornea, it is unlikely that there was, at any rate, a noticeable degree of microcornea in either of them, and as both went abroad they could not be re-examined. The first case, II 1, age 65 (1914), was always near sighted. With her right eye she read letters of 20 J; there was high myopia, diffuse lens opacity, and normal tension; left eye had perception of light, iridodonesis, a lens dislocated into lower part of vitreous chamber, a large posterior staphyloma of complete choroidal atrophy, much retinal pigmentation at periphery of fundus, with tension+1; optic disc of good colour and not cupped. Her son, III 7, age 24 (1910), had in his right eye myopia 22 D., V. with correction=6/36; in the left eye myopia 18 D., V. with correction=6/18; in each fundus were extensive myopic changes.—II 5, died in childhood, III 9, and 10, killed in the war. III 11, newly married. III 18, and 19, unmarried. III 27, two miscarriages. III 20, wrote that he and his daughter, IV 19, had perfectly sound eyes and that they had had no trouble with their sight. IV 20, and 21, in South Africa, both see well. IV 1, died aged three.

A LIST OF PUBLICATIONS THAT SHOW INHERITANCE OF MICROPHTHALMIA

1. **Pönitz.**—*Zeitschr. für Natur und Heilk.*, herausgegeben von den Professoren der chir. med. Academie zu Dresden. Dresden, 1822, Vol. VIII, Part ii, p. 60 (Ref. Gescheit, v. Ammon's *Zeitschr. für die Ophthal.*, Vol. II, p. 257, Dresden, 1832).
2. **Fischer, C. E.**—Zweier an Microphthalmus leidender Geschwister. Auszüge aus den Jahrbüchern der Krankheiten Lüneburgs, S. Hufeland's *Jl. der pract. Heilk.* Supplem. Heft. des Jahrg. 1827, p. 27. (Ref. Gescheit, Ant.) *Zeitschr. für die Ophthal.*, Vol. II, p. 257, Dresden, 1832).
3. **Wutzer.**—In Meckel's *Arch. f. Anat. u. Physiologie*, 1830, Parts 2. 3, p. 179). Quoted by Karl Himly in his "Die Krankheiten und Missbildungen des menschlichen Auges," Part i, p. 528. Berlin, 1843, and by Gescheit, Ant. *Zeitschr. für die Ophthal.*, Vol. II, p. 257, Dresden, 1832.
4. **Gescheit, Ant.**—"Ueber Microphthalmos oder der angeborene Kleinheit der Augen." v. Ammon's *Zeitschr. für die Ophthal.*, Vol II, p. 257, Dresden, 1832. (The two microphthalmic brothers mentioned by v. Ammon, F. A., in "Klinische Darstellungen der Krankheiten des Menschlichen Auges," Part iii, Table iii, Figs. 1 and 3, Berlin, 1841, are the same individuals as those described by Gescheit).
5. **Himly, K.**—"Die Krankheiten und Missbildungen des menschlichen Auges," Part i, p. 530, Berlin, 1843.

6. **Stoeber.**—*Gazette med. de Strasbourg.* Quoted by De Beck, ref. Loeb, Clarence. "Hereditary Blindness and its Prevention," *Ann. of Ophthal.*, No. 2, p. 245, Vol. XVIII, 1909, also, by Cunier, *Ann. d'Oculistique*, Vol. XIII, p. 33, Bruxelles, 1845.
7. **Cunier, M. F.**—"Microphtalmie et surdi-mutité héréditaires," *Ann. d'Oculistique*, Vol. XIII, p. 30, Bruxelles, 1845. (2 pedigrees).
8. **White Cooper.**—"Microphthalmos." *R.L.O.H. Reports*, Vol. I, p. 110, 1858.
9. **Harlan, Geo. C.**—"Report of an examination of the eyes of 167 inmates of the Pennsylvania Institution for the Instruction of the Blind." *Amer. Jl. of the Med. Sciences*, Vol. LXV, p. 410, 1873.
10. **Page, H.** "Transmission through three generations of microphthalmos, irideremia and nystagmus." *Lancet*, p. 193, August 8, 1874.
11. **Krükow.**—"Zwei Fälle von angeborenem Hornhautstaphylom," *Arch. für Ophthal.*, Vol. XXI, Part ii, p. 213, Berlin, 1875.
12. **Benson, A. H.**—"Congenital irideremia of both eyes." *Brit. Med. Jl.*, Vol. II, p. 359, 1878.
13. **Ravá, H.**—"Estrazione doppia di cataratta felicemente esequita." *Annali di Ottalm*, Vol. IX, Parts iii and iv, p. 281, ref. Nagel's *Jahresbericht*, p. 387, 1880.
14. **Manz, W.**—"Zwei Fälle von Mikrophthalmus congenitus nebst Bemerkungen über die zystoide Degenerationen des fötalen Bulbus." *Arch. f. Ophthal.*, Vol. XXVI, p. 154, Berlin, 1880.
15. **Schaumberg, C. F.**—"Casuistische Beiträge zu den Missbildungen des Auges." Inaug. Dissert. Marburg, 1882, ref. Nagel's *Jahresbericht*, p. 257, 1882.
16. **Mayerhausen, G.** "Directe Vererbung von beiderseitigem Microphthalmus." *Centralbl. f prakt. Augenheilk.*, p. 97, 1882.
17. **Schenkl.** — "Jahresbericht der deutschen Poliklinik in Prag," ref. Nagel's *Jahresbericht*, p. 281, 1884.
18. **Appenzeller.**—"Beitrag zur Lehre von der Erblichkeit des grauen Stars." *Mitteil. d. ophth. Klinik in Tübingen*, Vol. II, Part i, p. 120, ref. Nagel's *Jahresbericht*, p. 297, 1884.
19. **Pflüger, E.**—"Microcephalie und Microphthalmie." *Arch. für Augenheilk.*, Vol. XIV, p. 1, Wiesbaden, 1885.
20. **Magnus, H.**—Die Jugend-Blindheit, p. 36, Wiesbaden, 1886.
21. **Martin, F.**—"Ueber Microphthalmus." Inaug. Dissert. Erlangen, 1888, ref. Nagel's *Jahresbericht*, p. 227, 1888.
22. **Smith, Priestley.**—"On an instance of Hereditary Glaucoma and its cause." *Ophthalmic Review*, Vol. XIII, p. 215, 1894.
23. **Hirschberg, J.**—"Angeborener grauer Star als Familienübel," *Centralbl. f. prakt. Augenheilk.*, p. 271, 1897.
24. **Reber, M.**—"Mikrophtalmie compliquée d'hypermetropie excessive et d'anomalies de la macula, observé chez trois sœurs." *Ann. d'Oculistique*, Vol. CXIX, p. 445, 1898.
25. **Bruns, Henry Dickson.**—"A Microphthalmic Family." *Amer. Jl. of Ophthal.*, Vol. XVI, pp. 68-70, 1899.
26. **Scherenberg, K.**—"Beiträge zur Lehre vom reinen Mikrophthalmus." Inaug. Dissert Tübingen. 1900, ref. Nagel's *Jahresbericht*, p. 297, 1900.
27. **Lavagna, J.**—"Contribution à la tératologie et à l'embryologie oculaire." (Société méd. de Monaco, 17 avril, 1901). Nice; "Petit Niçois," ref. Nagel's *Jahresbericht*, p. 270, 1901.
28. **Barkan, A.**—"Congenital arrested development in the fundus of each eye." (San Francisco Society of Eye, Ear, Nose and Throat Surgeons.) *Ophthal Record*, Vol. X, p. 216, 1901.
29. **Argyll Robertson.** — Discussion, Ophth. Society, on Bronner's paper on Microphthalmos. *Trans. Ophthal. Soc.*, Vol. XXII, p. 211, 1902.
30. **Fuchs, E.** — Discussion, Ophthal. Society, on Bronner's paper on Microphthalmos. *Trans. Ophthal. Soc*, Vol. XXII, p. 212, 1902.
31. **Bronner, Adolph.** — "Notes on two families with bilateral congenital microphthalmos and cataract." *Trans. Ophthal. Soc.*, Vol. XXII, p. 209, 1902. (2 pedigrees).
32. **Osterroht, Dr.**—"Ein Fall von hochgradiger Hypermetropie bei angeborenem Mikrophthalmus mit inneren Komplikationen." *Beiträge zur Augenheilk.*, Deutschmann, Vol. VI, 60, p. 33-41, 1904.

33. **Sulzer, M.**—"Microphtalmie unilatérale droite." *Recueil d'Ophtalmologie*, p. 356, 1906.
34. **Harman, N. Bishop.**—"New pedigrees of cataract—posterior polar, anterior polar and microphthalmia, and lamellar." *Trans. Ophth. Soc.*, Vol. XXIX, p. 296, 1909.
35. **Harman, N. Bishop.**—"Ten pedigrees of congenital and infantile cataract; lamellar, coralliform, discoid, and posterior polar with microphthalmia." *Trans. Ophth. Soc.*, Vol. XXX, p. 251, 1910.
36. **Stuelp, O.** — "Ueber familiären Mikrophthalmus congenitus bei 8 von 14 Geschwistern." *Arch. f. Ophthal.*, Vol. LXXXVI, p. 136, 1913.
37. **Thomsen, Chr.**—"Ueber die Vererbung des Mikrophthalmus mit und ohne Katarakt." Inaugural - Dissertation. Rostock, 1913.
38. **Ischreyt, G.**—"Zur Kasuistik der Missbildungen des Auges. Mikrophthalmus." *Klin. Monatsbl. f. Augenheilk.*, Vol. LVII, July-December, p. 504, 1916.
39. **Ring, G. O.**—"Concerning coloboma of the iris in association with congenital cataract." *Ophthalmic Record*, March, 1917, ref. *Arch. of Ophthalm.*, Vol. XLVI, p. 392, 1917.

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A LETTER FROM BENJAMIN GIBSON OF MANCHESTER ON THE SUBJECT OF THE PUBLICATION OF HIS BOOK

BY

R. R. JAMES

LONDON.

THE following letter, which has recently come into my hands, may be of interest to readers of the BRITISH JOURNAL OF OPHTHALMOLOGY. Addressed to Messrs. Cadell & Davies, Booksellers, London, it runs as follows:—

Manchester, *October 22*, 1810.

GENTLEMEN,

By the recommendation of my friend Dr. Ferriar, I address you,

respecting a small volume, which I am about to publish and which is now ready for the press. The subject of it is surgical: consisting of several of the finer operations upon the eye, which, as far as I know, have not until now been laid before the public; the knowledge of them being confined to a few of my surgical friends. For the merits of the work I refer you to Dr. Ferriar, or to Dr. Henry of this place, who have both read the manuscript. With respect to its length, I think that it will extend to about 140 pages or more, printed of the same size as Dr. Ferriar's Medical Histories. I intend to have it printed at Warrington, that I may correct the press myself. Attached to the work there will be two plates; one of instruments in outline only. The other, which consists of four drawings of eyes, may either be executed in the usual style of engraving, or be coloured, whichever you most approve of; provided you chuse to publish the work.

And now, Gentlemen, with respect to terms: I do not wish to dispose of the copyright, nor do I publish for any pecuniary profit. If, therefore, after making proper enquiries of the merits of the performance, you are willing to take the whole expense upon yourselves and allow me thirty copies (many of which will be given where the book would not have been bought) you are welcome to print any kind of edition of it you please, and receive any profits arising from its sale. I shall be glad to receive as early an answer as your leisure will permit; and if I have omitted any points of information, upon a business I am unacquainted with, I shall be ready to add them.

I remain, Gentlemen,
Yours very respectfully,
B. GIBSON.

This letter is endorsed on the back, as follows:—

London, *October* 26, 1810.

SIR,

We are favoured with your obliging letter, and are very ready to undertake the publication in question, on the terms proposed. You will be so kind then as to direct the printers (Mr. Haddock, probably) to print 500 copies, in a manner similar to that of Dr. Ferriar's new edition; and, with regard to the plates, we request the engravings may be executed in whatever way you may judge best.

We are, Sirs,
Your obliged and very obedient servants.

The book in question is the "Practical Observations on the formation of an Artificial Pupil, with remarks on the extraction of Soft Cataract, through a puncture in the Cornea," by B. Gibson. 8vo. London, 1811. It was printed by Haddock, of Warrington,

and there is a copy in the Bowman Library, a presentation copy from the author to James Wardrop.

Benjamin Gibson was born at Newcastle-on-Tyne in 1784. After having received his general education at Richmond, Yorkshire, he was apprenticed to Mr. Ingham, surgeon, of Newcastle-on-Tyne, and in 1796, he was in London, studying under Mathew Baillie. He went to Manchester, as assistant to the celebrated surgeon, Charles White, in 1799. Gibson was surgeon to the Manchester Infirmary, 1804-1812; he was also assistant surgeon to the Royal Manchester and Salford Volunteer Regiment of Infantry. He suffered from consumption, and died at Ardwick, March 6, 1812. These few facts about him are extracted from Brockbank's "Honorary Medical Staff of the Manchester Infirmary, 1752-1830."

CONGENITAL ABSENCE OF THE LENS, WITH SPECIAL REFERENCE TO AN APHAKIC HUMAN EMBYRO*

BY

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CONGENITAL absence of the lens is of such rare occurrence that some observers have denied that it is ever found apart from gross malformations such as anophthalmos and severe degrees of microphthalmos. From time to time, however, cases have been reported clinically, but have not been verified by microscopic examination. All these cases, as far as I am aware, occurred in America, and the first was reported by Baker in 1887. The details in this case are so meagre that very little information can be gained from the account. Since then other cases have been reported by Cherryholmes (1902), Adams, Hardy and Sherer (1915). In Cherryholmes' case there was microphthalmos with an interpupillary distance of only 1⅞ inch, and also nystagmus, convergent strabismus and amblyopia.

Cases of microphthalmos which have been subjected to microscopic examination are many, and in the majority of them (for example, Wiegel's case of microphthalmos and atypical vitreous) the lens could be identified, though it was often only represented by a few degenerate epithelial cells enclosed in a much wrinkled capsule. Cases in which it was entirely absent were usually those severe degrees of failure of development in which all that could be found of the eye was a cyst or vesicle of entirely atypical structure, or

* Read on May 13, 1921, before the Section of Ophthalmology of the Royal Society of Medicine.

possibly showing some traces of retina, but without lens, ciliary body, iris or pupil, as in the case described by Becker. The question of the possibility of congenital aphakia occurring in an otherwise normal or nearly normal eye can therefore still be said to be an open one.

As to aphakia being seen in an embryo, one case has been described by Rabl in an Amblyostoma embryo. In this instance the condition was unilateral and was associated with general failure of ectodermal structures on that side of the head, the central nervous system and the optic cup being smaller than on the other side and the whole head deformed. Cases of simple failure of development of the lens with a normal or almost normal optic cup have been hitherto unknown, and the human embryo about to be described forms, as far as I am aware, an unique case and goes far to prove that aphakia can occur as an isolated malformation at any rate in foetal life.

As is well known, the human lens normally develops from an invagination of the whole thickness of that portion of the surface ectoderm with which the primary optic vesicle comes in contact. The following table* shows the normal correlation between the degrees of development of the eye and the size of the embryo.

Length	No. of Somites	Condition of optic cup	Condition of lens
4 mm.	35	Distal wall of optic vesicle thickened and almost in contact with surface ectoderm. Wide communication between optic vesicle and cavity of ventricle	Rudiment of lens recognizable as thickened epithelial plate
6.75 mm.	38	Optic cup formed. No retinal pigment yet. Stalk of optic cup still very wide	Rudiment of lens a flat groove in which are cell proliferations
7 mm.	40	Traces of pigment in retina	Lens vesicle just closed, but not separated from ectoderm. Proximal wall thickened
13 mm.	—	Choroidal fissure almost closed. Abundant retinal pigment. Anterior chamber not yet formed. No nerve fibres in optic nerve	Lens vesicle closed and separated from ectoderm by mesodermal cells. Lens fibres beginning to appear

The aphakic human embryo (one of Professor Frazer's collection) which forms the subject of this paper was compared with a normal human embryo of 13 mm. (measured when fresh). After sectioning, the aphakic embryo measured 8·64 mm. and the normal one 8·67 mm. They are thus of approximately the same size. They also show the same degree of general development as evidenced by

* Modified from the "Normentafel" of Keibel and Elze.

the condition of the central nervous system, of the submaxillary outgrowth (which is just appearing in both) and of the nasal fossae. We can thus assume that the aphakic embryo had reached a stage of development normal for its size. No sign of failure of any ectodermal structure other than the lens was discovered.

The eyes of this embryo will now be described in detail. As far as one can judge the condition is similar in both eyes, but some of the serial sections passing through the left eye are broken. The drawings and measurements were therefore made from the right eye.

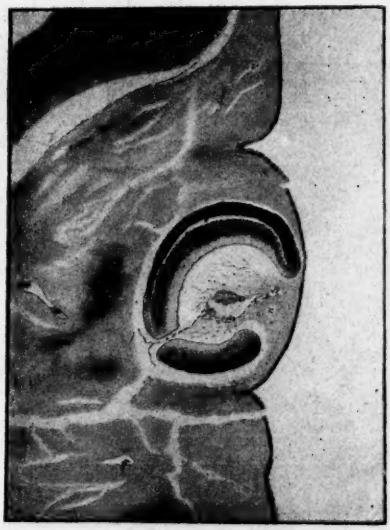

FIG. 1.

Section through right eye of aphakic embryo. Passes through upper part of fissure. Shows hyaloid vessel, absence of lens, inner and outer layers of optic cup, etc.

The optic cup occupies its normal position on the side of the head. It is well formed, roughly spherical in shape and measures 0·51 mm. in diameter. (The right optic cup of the comparison normal embryo measures 0.54 mm. in diameter.) The choroidal fissure is still open with the lips of the fissure almost in apposition. The fissure, however, does not extend into the optic stalk. (The choroidal fissure of the comparison embryo is closed except for a notch at the pupillary margin.) The hyaloid vessel enters the globe at the extreme upper end of the fissure and there is a larger vessel than normal running in the line of the fissure outside the optic cup. The blood drainage

is that usual at this stage, namely by two main systems, both joining the cavernous sinus, one *viâ* the supraorbital plexus and the other by broken channels *viâ* the remainder of the anterior plexus. The hyaloid vessel is large and full of blood and breaks up into numerous blood spaces in the pupil. These drain round the edges of the pupil into the large vessel running in the line of the cleft and into other scattered channels in the surrounding mesoderm. This can be seen in Figs. 1 and 2. The condition of the retina, both inner and pigment layers, is normal. This can be seen by comparison of Figs. 1 and 2 with Fig. 3, which shows the eye of the normal 13 mm. embryo for comparison. It is to be noted that in the normal embryo the anterior chamber has not yet been

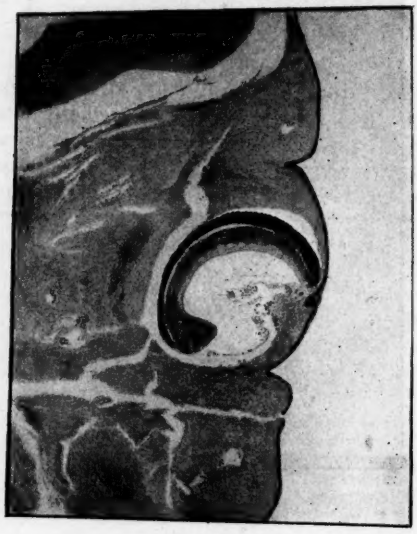

FIG. 2.

Section through right eye of aphakic embryo, slightly lower in the series than Fig. 1. Shows in addition abortive lens as epithelial projection from deep surface of corneal ectoderm.

formed, there being still mesoderm situated deep to the cornea and spreading round the lens on each side. In the abnormal embryo there is no trace of a lens beyond the presence of a very small blunt-pointed epithelial projection from the inner side of the surface ectoderm. This is almost in the middle of the pupil (namely 0·01 mm. below and slightly anterior to the middle). It is approximately 0·02 mm. thick and affects the deep layer of the ectoderm only and is not accompanied by a corresponding

depression on the surface. It possibly represents an abortive attempt at a lens thickening which has never invaginated or separated. (It is shown in Fig. 2.) Surrounding the hyaloid vessel is a loose reticular tissue showing fibrils similar to those seen in the developing vitreous in the 13 mm. embryo. The mesoderm deep to the cornea passes round the pupillary margin and blends with the mesoderm surrounding the vessel, as in the normal embryo.

The condition then is one of congenital aphakia. It is of course merely speculation to consider the possible end result had the embryo continued to develop. The somewhat delayed closure of

FIG. 3.

Section through eye of normal 13 mm. embryo. Shews similar stage of development of optic cup and also a well formed lens vesicle separated from surface ectoderm.

the foetal cleft suggests that some form of coloboma might have arisen, but it is interesting to note that there is no sign of microphthalmos at present, the eye being of normal size for the stage of development of the embryo. Some observers (e.g., Bach) consider that microphthalmos is produced by gross abnormality or absence of the lens, but this is rendered doubtful both by observations showing that the optic cup is self-differentiating, and also by the work of Ochi on the experimental production of abnormalities of the head in chicks by alterations in the osmotic pressure in the albumen in the neighbourhood of the head. He

succeeded in producing microphthalmos, but the small eyes nearly always contained a lens.

The possibility that in this embryo a lens did form and subsequently degenerated has been considered, but the most careful examination has failed to reveal any traces of it. Also W. H. Lewis has shown that degeneration of the lens after it has once formed is usually associated with overgrowth of mesoderm between the optic cup and the lens causing their separation sooner than usual, and in this case there is no evidence of such abnormal growth.

The case is also of interest in that it offers confirmatory evidence concerning the experimental results of W. H. Lewis, Spemann and others working on the power of self-differentiation possessed by various organs. Both Spemann and Lewis have shown that invagination of the optic vesicle to form the optic cup is not dependent either on the formation of a lens or even on contact with the surface ectoderm, since it occurs in optic vesicles transplanted into various parts of the embryo. Lewis, however, states that when an optic cup forms from a transplanted optic vesicle without a lens, the invagination is not perfect and a small irregular cavity is formed which is filled with mesenchyme, there being no trace of the formation of the vitreous or of the chambers of the eye. This may well be so under experimental conditions, and may possibly be accounted for by distortion of the optic vesicle at the time of the operation and by the abnormality of the blood supply in such a transplanted vesicle. The present case shows that in aphakia arising without external interference the vitreous at any rate begins to differentiate quite normally.

Though the invagination of the optic cup is independent of the presence of a lens, it is generally accepted that the formation of a lens is dependent on contact between the developing optic cup and the surface ectoderm. The exact position of contact appears to be immaterial. A chemical substance has been postulated which is secreted by the optic cup, which substance, acting during a certain period, stimulates the overlying ectoderm to proliferation and lens formation. In this embryo contact has undoubtedly taken place, since there is a trace of ectodermal thickening, but one might suppose that there had then been failure of the continued stimulus (chemical or otherwise) necessary to produce complete differentiation of the lens. It is conceivable that this failure on the part of the optic cup to produce the requisite stimulus need not imply sufficient abnormality in the cup to prevent its own differentiation.

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THE YIELDING OF THE OPTIC NERVE-HEAD IN GLAUCOMA

BY

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LONDON

IT is not an uncommon thing to find writers on glaucoma, making a casual reference to the possible influence on their subject of anatomical variations in the intraocular portion of the optic nerve, and in the canal which contains it. Such references are, however, for the most part so vague and indefinite as to provoke the suggestion that they are not founded on well-ascertained data. The writer has felt the need of greater precision in our knowledge of the subject, and this has been especially impressed upon him in connection with his study of the various paracentral scotomata which are met with in the glaucomatous. Of the various sources of information to which he is indebted, he would wish to make an early and special acknowledgment of two, viz., Salzmann's "Anatomy and Histology of the Human Eyeball" translated by Dr. E. V. L. Brown (University of Chicago Press, Chicago, 1912), and Fuchs' illuminating article "On the Lamina Cribrosa" (*Arch. f. Ophthal.*, 1916, Vol. XCI, Part iii). The latter paper is by no means an easy one to study in the original, and this for various reasons, not the least of which is the arrangement therein followed. The writer desires to express his acknowledgments to Mr. E. E. Henderson for the help he has derived from that gentleman's lucid abstract of this voluminous paper (B.J.O. 1918, Vol. II, p. 50), and for the personal help he has given him in unravelling difficult details of the translation. It is in no sense derogatory to this abstract to say that, clear and helpful as it is, it was only by careful study of the original, and by putting together facts and statements in the order of sequence in which they affect the problems the writer had before him, that a clear view of the subject was obtained. Glaucoma teems

with problems which demand and deserve very careful study. Such study cannot fail to add to our knowledge, and to help to dissipate the clouds which at present obscure our conception of this great subject. In the course of some work he had in hand, the writer had first to get his own ideas straightened out, so far as our present knowledge permits, and he has felt that having done so, his results may be helpful to others, as well as to himself.

Anatomical Considerations

It is essential, in the first place, to gain as clear a conception as is possible of the anatomical arrangements of the intraocular or bulbar portion of the optic nerve, of the canal in which it lies, and of the relationship of the nerve to the canal. The exact parts we are about to discuss will be understood, if we take a cleanly enucleated eye, with the optic nerve cut off flush with the scleral surface, and if we then make a study of the nerve and of the contiguous coats of the eye.

The Entrance Canal of the Optic Nerve

This is divided for anatomical purposes into a choroidal and a scleral portion. The inner or choroidal opening of the canal is

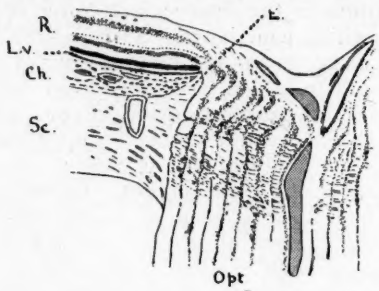

FIG. 1.—Semi-diagrammatic representation of lining of the canal of entrance of the optic nerve (modified from Salzmann). R retina, Ch choroid, L.v. lamina vitrea of choroid. The inturned selvedge-edge E of the scleral tissue, which lines the choroidal part of the canal, ceases at the lamina vitrea. Sc sclera, Opt. optic nerve. (E.C.I.E.)

about 1.5 mm. in width; it may be taken to be bounded by the margin of the opening in the choroid, and by the contiguous part of the sclera. The outer opening, or scleral foramen, as is well-known, is usually much wider than the inner; its boundary is formed by the outmost layers of the sclera.

The wall of the canal is formed by a whitish fibrous tissue, which, in the choroidal part of the tube, is clearly distinguishable from the uveal layers, but which, in the sclerotic portion, blends with the adjacent sclera, with which it appears to be continuous. It is as

though the sclera around the inner foramen ended in a selvedge edge, which is carried up clear to the membrane of Bruch; scleral tissue thus lines the whole of the optic canal (Fig. 1). There has been much dispute as to the exact nature of this lining, and for this reason Salzmann has preferred to describe it by the indefinite and non-committal name of "the border tissue." It is generally accepted that fibrous tissue elements from this border tissue extend into the framework of the optic nerve, and so help to form the cribriform plate. Differences of opinion exist (1) as to whether elements derived from the choroid take any part in the formation of the fibrous tissue framework of this part of the nerve, and (2) as to the exact part played by other tissues.

It is important to understand that the framework or scaffolding of this part of the nerve is derived from two sources: (1) the

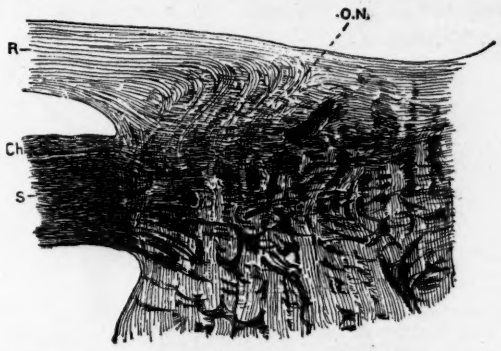

FIG. 3.—The transverse lamellae lie mainly in the anterior part of the optic canal, and are not very strong, as compared with the glial tissue which lies in bundles along the length of the nerve.

mesodermal, which we have just been discussing, and (2) the ectodermal. The latter tissue is usually spoken of as "neuroglia" or "glia." Considerable differences of opinion exist as to the exact relationships of these two sets of tissue within the intraocular portion of the optic nerve. Fuchs, who has recently conducted an exhaustive study of the subject, holds that there is a close interconnection between the connective tissue and the neuroglia. In spite (a) of their differences of origin, and (b) of the fact that staining reactions show that there are equally profound differences in their chemical nature, he finds that anatomically they are closely intermingled, and that the limits between them are not well defined. These views conflict with those of earlier writers. Such discussions are to a large extent academic. It is, however, of much greater and of more practical interest to note that the supporting tissue found in this portion of the nerve varies very greatly both in its

distribution and in its amount. Thus, according to Fuchs, we find in some cases a tendency for a large amount of this tissue to lie in bundles along the direction of the length of the nerve, and so to run from before backwards, whilst in others it consists of sheets of laminae, which cross the nerve trunk at right angles to its course and which may and often do, run clean across all round from the marginal ring of the canal of the optic nerve to the central vessels. Moreover, these lamellae may vary in number, in strength and in completeness. These points are well illustrated by a comparison of Figs. 2, 3 and 4. In Fig. 2 it will be observed that the large number of strong lamellae, superposed upon each other, give the lamina cribrosa a considerable depth as measured from before backward, whilst in Figs. 3 and 4 the paucity of lamellae has diminished the depth of that membrane. Nor is this all that can be learnt from these illustrations. The strength of the individual lamellae, apart altogether from their numbers is obviously greater in

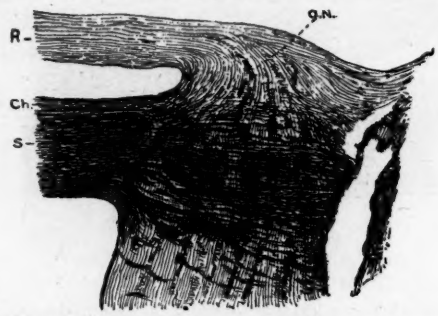

FIG. 2.—The transverse lamellae are here very strong and are also very numerous. The result is that they build up a thick strong lamina cribrosa. The longitudinally placed glial elements are relatively poorly marked in this drawing.

the nerve depicted in Fig. 2 than in those of Figs. 3 and 4. Still one more point, the lamellae are not always of even strength right across the section of the nerve; indeed they appear to vary widely in this respect. If one may illustrate the point: In one case the membrane may be attached to the canal all round its circumference and may stretch across the whole section of the nerve; in another, only a part of the membrane may be present, the remainder being wanting or weak. Granted then such differences in the number, thickness, and completeness of the individual lamellae, it must be at once obvious that not only will there be great differences in the resistance to pressure of the laminae cribrosae thus built up in different eyes, but also that similar differences will surely be found in different parts of this membrane, even in the same eye. Fuchs believes that those laminae cribrosae, whose elements are mainly

made up of transversely running lamellae (*Fig. 2*) must be much more resistant to pressure applied, as intraocular pressure always must be, along the axis of the nerve, than are those in which the bulk of the tissue is built up of bundle-like elements, which run in the direction of the long axis of the nerve (Fig. 3). This proposition is self-evident, for the transverse lamellae, tethered as they are to the sides of the canal of the optic nerve, will check even the slightest tendency to a push of the nerve directly backwards, whilst the bundles, which run in the axis of the nerve, will readily be displaced, along with the structures between which they lie.

There is another point, of which we are not ignorant, but which has been emphasized for us by Fuchs in his paper under reference, viz., that the situation of the lamina cribrosa varies greatly in different

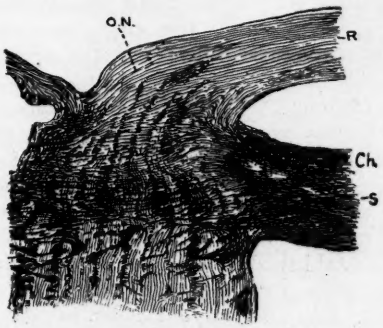

FIG. 4.—This resembles *Fig. 3*, except that the transverse lamellae lie mainly in the posterior part of the canal (the usual arrangement) and the longitudinally placed glial bundles are not quite as strongly in evidence.

FIGS. 2, 3 and 4 are diagrammatic representations, constructed from Fuchs' microphotographs, and modified in accordance with the text of his article to make the points he emphasizes clearer. They have been kindly supplied by the Oxford Medical Publications. The lettering is the same in all. R, retina; Ch. choroid; S, sclera; O.N., optic nerve.

eyes. This of course depends (1) on the position at which the various connective tissue lamellae, which between them, form the lamina, find their attachment to the walls of the optic canal and (2) on the number of these lamellae. Figures *2*, *3* and *4* illustrate this point clearly. For convenience sake, we shall take them in reverse order. In Fig. 4, the lamina cribrosa lies in what is usually described as its normal position, i.e., in the posterior segment of the canal. In Fig. 3 it lies almost wholly in the anterior portion of the canal. Figure *2* forms a contrast with both of the preceding ones, since in it the lamina appears to occupy practically the whole length of the canal.

It is obvious, as Fuchs has pointed out, that these variations in anatomical structure must greatly influence the ophthalmoscopic

appearances presented by different cases of optic atrophy, unaccompanied by increase of pressure; in the first case taken above (Fig. 4), the cup would be much deeper than in either of the others (Figs. 2 and 3), that is, if we assume that the underpressed anterior surface of the lamina cribrosa forms the floor of the cup.

Even under conditions in which pressure plays a part, the effect on the lamina is not quite so simple, according to Fuchs, as we have always been led to suppose. In the earlier stages, the whole of this supporting framework is not affected; for it is the glial tissue which yields first to pressure and disappears, whilst the connective tissue lamellae of mesodermal origin are still able to hold their ground. It is the glial fibres in the anterior part of the nerve head which are first destroyed, and their destruction, according to Fuchs, actually precedes that of the nerve fibres themselves. Later still, the glial elements of the whole framework of the intraocular portion of the nerve disappear. All these changes may occur before any displacement of the lamina cribrosa is in evidence. If, however, intraocular pressure is continued, the lamina yields as a whole, being pushed back into the optic nerve trunk, which is thereby proportionally shortened, and at the same time made thicker. Part of this thickening is due to changes in the nerve fibres, and to the development within the nerve of a condition which resembles a profuse oedema. Later, when atrophy is established, the nerve becomes thinner than before.

It remains to state, in support of what has been put forward above, that Fuchs has notes of four cases, in which the sectioned papillae showed depressions of their surfaces varying from 0·45 to 0·73 mm.; in none of these cases was the lamina markedly displaced, the cupping being due to destruction of the glial tissue and of the nerve substance. It is both noteworthy and strange that no evidence could be discovered in these cases of any increase, either in the strength or thickness of the lamina, such as might explain its remaining in position; indeed, in some of them, it appeared to be thinner than normal.

The sequence of events would appear to be as follows: (1) The disappearance of the delicate anterior glial fibres; (2) the disappearance of the deeper glial fibres incorporated in the lamina, or their fusion under pressure with the connective tissue lamellae; (3) the bending backward of the connective tissue lamellae, which build up the lamina; (4) the compression, sclerosis and even thickening of the lamina probably as a result of the load it has to bear; and finally (5) the thinning and atrophy of the lamina as a result of continued pressure; at this stage, not only do the individual fibres become thinned and even disappear, but also a formation of spaces occurs, the lamina being thus increasingly broken up.

Side by side with these changes in the supporting framework of the intraocular portion of the nerve, we have to take account of what is happening to the nerve fibres themselves. This is the more necessary because the course of events appears to differ widely in different cases, even so far as the nerve elements themselves are concerned. Thus, we may find extensive atrophy of the nervous tissues at a stage when only the anterior glial fibres have disappeared, and when the connective tissue lamellae, and consequently the lamina which they build up, are still in a normal position. Under such circumstances, as Fuchs points out, the picture presented might be that of an increased physiological cup with nerve atrophy. We have but to remember how deep the connective tissue lamellae may lie from the surface of the papilla in certain cases, and to cast our minds back for a moment to Fuchs' observation of cups up to nearly 0.75 mm. in depth occurring under these conditions, to realize how difficult the ophthalmoscopic diagnosis of the exact condition present may easily become. On the other hand, in some cases, the lamina may yield at an early stage, whilst the nerve fibres are still intact. From the point of view of the symptoms presented, the cases will vary greatly. On the one hand, there may be comparatively shallow cupping with marked atrophy and early loss of vision; on the other, deep cupping with very moderate atrophy and comparatively little impairment, either of central or peripheral sight.

If we may accept Fuchs' work—as there is every reason to believe we may, not only on account of its author's reputation, but also because it falls into line with our clinical experience of increased intraocular pressure, with all its variations in the signs and symptoms present—we are bound to agree with its author that the problem of glaucoma is not the simple one of the physical effects of pressure alone. No one who has studied the congeries of conditions which we classify under the term "glaucoma" can fail to be convinced of the depth of our ignorance on the subject we are discussing. This admission, however, implies neither sympathy nor agreement with the views that sometimes find expression in medical literature, to the effect that the symptoms of glaucoma are independent of a rise in intraocular pressure. Such an idea is little likely to be accepted by anyone who has studied the subject and who takes broad, balanced views of the situation. Unfortunately, the study of glaucoma has, perhaps more than any other domain of ophthalmology, been invaded by theories, which have had little to recommend them, and which have only served to add confusion to the subject. Fortunately, their influence has generally been ephemeral, though it can hardly be said to have been harmless.

There are many departments of the study of glaucoma, which will

be illumined by advances in our knowledge of the anatomical peculiarities that may be met with in the intraocular portion of the optic nerve and of the canal in which it lies, as well as of the relationships of the one to the other. Mr. A. Hugh Thompson read before the Oxford Ophthalmological Congress of July, 1920, an extremely interesting paper on "Physiological and Glaucoma Cups," pointing out how difficult it is in certain cases to draw a clear boundary line between physiological and pathological cupping. More recently, Mr. R. Pickard has taken up the same theme in a paper presented to the Royal Society of Medicine on January 14, 1921. Both these communications deal with the subject from the clinical point of view, and it is to be hoped and anticipated that they will stimulate others to carry the work still further. It is obvious that the most hopeful line of progress will lie in a combination of clinical and pathological investigations, for we cannot interpret aright what we see with the ophthalmoscope unless we check and simplify it by what we learn in the pathological laboratory.

Again, much interest has centred in recent years on the information furnished to the student of glaucoma by the results of careful perimetry and scotometry. Roenne's step, Seidel's sign, Bjerrum's sign and the jagged scotoma to which attention has been drawn by the writer, probably all find their explanation in (1) injuries to the nerve bundles as they pass over the edge of the disc, and (2) interference with the circulatory supply of the injured fibres in particular, and with that of the retina at large. How is the injury inflicted? Why should one mass of fibres—those, for instance, that emerge from a definite and circumscribed area above or below the disc—be more injured than any others? Why should we find the characteristic scotoma of glaucoma continuous as a rule with the blind spot, whilst exceptionally, but by no means rarely, the paracentral scotoma observed may be separated from that spot by large areas of clear vision? Why should the ophthalmoscopic picture of the glaucoma cup vary so very widely in the overhanging of the scleral edge, and in the kinking of the vessels, not only in different cups, but also in different sectors of the same cup? Why should the scleral edge of the inner foramen as seen in microscopic sections, seem at times so much sharper than at others, carrying with this peculiarity the suggestion that its power for harm, alike on the nerve fibres that flow over it, and on the vessels which accompany them, varies greatly in different cases? Are we here dealing with variations in the normal distribution of the retinal nerve fibres? Or, have we to do with uneven yielding of one or another of the two elements of the connective tissue that makes up the lamina? Or, is it a question of that direct pressure on the nerve fibres themselves to which Fuchs has drawn attention in this valuable paper? Or, must we explain the phenomena by a combination of all these influences?

Finally, is there in glaucoma some dark sinister factor which pathology has so far failed to grasp, some nameless, hitherto shadowy, influence for evil, whose detection has evaded all the means at our disposal?

These are very difficult questions, but not less attractive and important on that account. Nothing will do more to hinder the advance of our knowledge of glaucoma than the assumption that we really understand the subject, and probably nothing will be more likely to lead to progress than a candid exposure of the depth of our ignorance. For these reasons the writer does not hesitate to ask such questions as the above without making any serious attempt for the present to provide an answer to them.

SLOUGHING CORNEA IN GRAVES'S DISEASE AND IN DISSEMINATED SCLEROSIS

BY

T. HARRISON BUTLER

LEAMINGTON SPA.

ARNOLD KNAPP contributed to the *Archives of Ophthalmology* for March, 1918, a paper upon the loss of the cornea in Graves's disease. This catastrophe is not, he says, so very uncommon, and he recounts a case in which he succeeded in saving useful vision in one eye. In the following case first one cornea and then the other sloughed away, and ultimately the patient died.

On February 7, 1918, Mrs. P., aged 40, was sent to consult me by Dr. Bury, of Leamington Spa, who had tried every known treatment to relieve the symptoms of an aggravated case of exophthalmic goitre. On examination, in addition to all the usual signs of the disease, I found that there was an exaggerated proptosis of each globe, so much indeed that, when the eyes were closed as much as possible the left cornea was exposed. I strongly advised Mrs. P. to let me suture the left lids together. She was unwilling to follow this advice, and I did not see her again till March 2, 1918, when I examined her in bed at her own home. I found intense chemosis and brawny swelling of the conjunctiva of both eyes: the proptosis was even more pronounced: and the left cornea was infiltrated in its lower quadrant, and was anaesthetic. The tension of both eyes appeared to be raised, but there was nothing in their appearance to suggest glaucoma. I concluded that the apparent hardness was due to the tense state of the tissues behind the eye. Subsequent examination with the Schiötz tonometer showed that the tension was 20. I admitted Mrs. P. to the Warneford Hospital, where we instilled atropin, and carefully bandaged the eye. On March 3 a definite ulcer had formed in the

lower portion of the left cornea. In spite of treatment the ulcer slowly spread, and onyx appeared. On March 7 I freely incised the swollen conjunctiva and performed a Guthrie's section upon the cornea. Pus escaped from the layers of the cornea. Incisions into the brawny conjunctiva did not diminish the swelling, and blood alone escaped from the cut surfaces. This treatment proved futile. On March 9 under ether I freely cauterized the ulcer and sutured the lids together. As recommended by Priestley Smith, each lid was split from canthus to canthus, the raw surfaces everted and sewn together. On March 11 the sutures all cut out and the eye reopened. On March 27 the ulcer which was still spreading was again cauterized and covered with a conjunctival flap. This flap failed to remain over the cornea. The cornea finally sloughed and the eye, which was intensely painful, was enucleated under ether.

On March 24, the right cornea which had never been anaesthetic, began to ulcerate. In spite of treatment the cornea soon sloughed away. The general condition of the patient was so bad that no active measures were adopted to save the second eye, and after another fortnight she died of heart failure.

It is noteworthy that after removal of the eye the conjunctiva remained brawny and turgid.

The disease was so severe that I doubt whether earlier treatment would have saved the eyes, nor indeed did there seem much chance that her life would be preserved. In spite of the rapid and feeble pulse, ether anaesthesia by the open method was well tolerated, and the two operations were not followed by any shock or aggravation of symptoms.

In January, 1919, I was consulted by Mrs. M., age 36. The history was that she was quite well two years ago when she noted that the left leg began to drag after a long walk. She was found to be suffering from disseminated sclerosis. Examination showed that her knee jerks were greatly exaggerated, that she had well developed ankle clonus, and that she had great difficulty in walking. The right eye began to water in September, 1918, and the lid drooped. In October the eye became inflamed. When I examined her the right eye was injected, the cornea was anaesthetic and was perforated in its lower aspect. The anterior chamber was three-quarters full of pus. The upper half of the cornea had a healthy appearance. Mrs. M. was admitted to the Coventry Hospital. In spite of treatment the cornea sloughed away. The left eye remained normal. The end of the case was tragic in its suddenness. One day Mrs. M. was peacefully eating her dinner sitting up in bed, when without warning she became cyanosed and convulsed. She died before it was fully realized that she was in any danger. A post-mortem examination showed no cause for her asphyxia, which

was apparently due to laryngeal spasm. I am informed that it is not unusual for patients with disseminated sclerosis to die in this manner. The loss of the eye was undoubtedly due to trophic changes probably caused by a sclerotic area in the nucleus of the fifth nerve. I am not aware that such a case has been described before, and it must be very unusual.

COUNCIL OF BRITISH OPHTHALMOLOGISTS

Report on the Standardization of the Notation of the Axes of Cylinders

The Council is of the opinion that the absence of any agreed standard for the notation of the axes of cylinders is liable to give rise to mistakes in the making up of spectacle prescriptions. It finds that it is the practice of most opticians in this country to transcribe all prescriptions into a definite standard notation before sending them into the workshop. The Council considers that it is undesirable that the necessity for this should exist, for it is beyond doubt that when these prescriptions fall into the hands of less careful opticians, many mistakes arise, which would be avoided if ophthalmic surgeons adopted a standard notation.

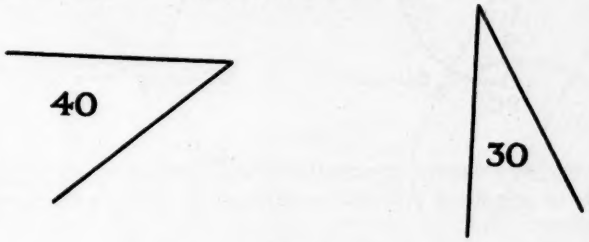

FIG. 1.

The methods of notation in use may be grouped as follows:

(A) That in which angles only up to 45° are used.

In this method, a diagram is drawn indicating the axis and the nearest horizontal or vertical meridian. The value of the angle separating the lines is written between them.

(B) Those in which angles up to 90° are used:—

(1) The horizontal meridian is taken as zero, and the angle is always read below. Thus "60° Down and In" means that the axis is separated from the horizontal by 60° and is pointing

downwards and inwards towards the nose. The other direction is described as "Down and Out," that is, downwards and outwards towards the cheek.

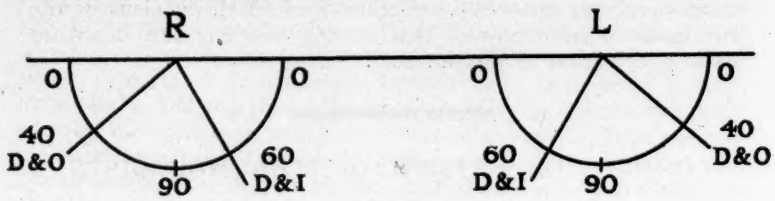

FIG. 2.

(2) The angle is reckoned from zero in the horizontal meridian on the nasal side of each eye, reading upwards or downwards to 90° in the vertical meridian. The direction is described as "40° Up and In," "60° Down and In," or more briefly, 40° Up, 60° Down."

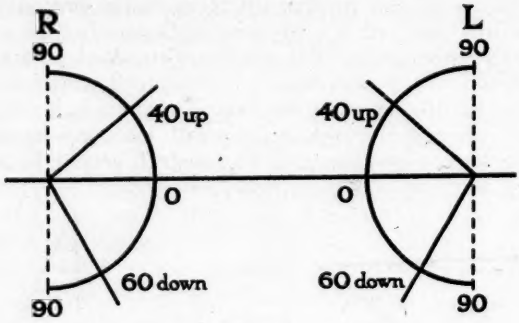

FIG. 3.

(3) Zero is at the lower end of the vertical meridian and the angle is measured inwards or outwards to 90° in the horizontal meridian.

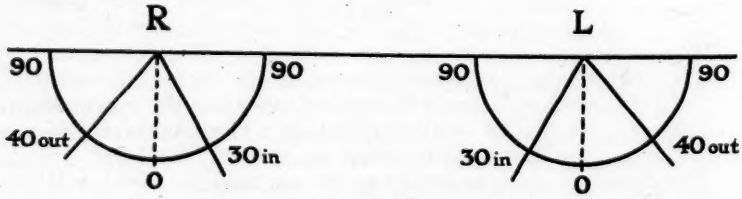

FIG. 4.

(C) Those in which angles up to 180° are used. There are four varieties, and in each the scale may be read below or above

the horizontal. In all, the zero lies in the horizontal meridian.

1 and 2. *Zero* lies at the temporal side of each eye, the scale being read above the horizontal in No. 1 and below in No. 2.

3 and 4. *Zero* lies at the nasal side of each eye, No. 3 being read above and No. 4 below.

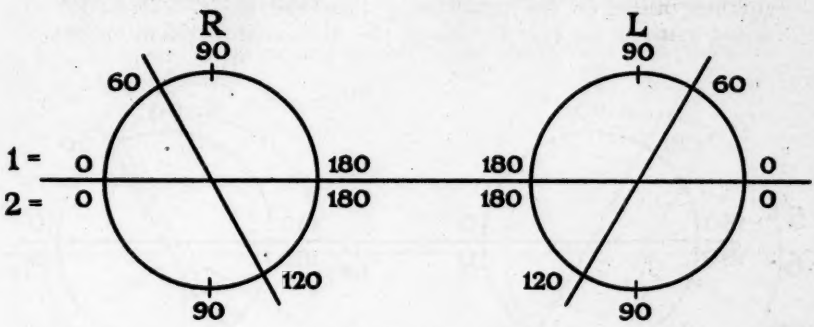

FIG. 5.

It will be observed that the numerical value obtained for the position of an axis is the same when employing either method 1 or 4, and similarly either method 2 or 3.

5 and 6. *Zero* lies at the right side of each eye from the point of view of the observer, No. 5 being read above and No. 6 below.

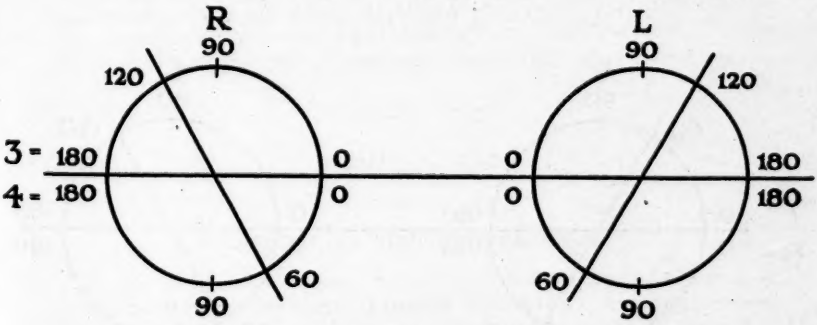

FIG. 6.

7 and 8. *Zero* lies at the left side of each eye, from the point of view of the observer, No. 7 being read above and No. 8 below.

Again it will be observed that the same numerical value will be obtained when using 5 as 8 and 6 as 7.

Every mathematical science adopts one and the same notation (commonly called the "Standard Notation") of positive angles, by

means of a radius vector starting from the horizontal on the right of its centre of rotation, and rotating counter-clockwise to an angle of 180° degrees on the left. If this Standard Notation were universally employed for indicating the position of the cylinder axis, there would be no need for an ophthalmic surgeon to put anything more on his prescription than the angle. Thus 45° would mean an angle of 45° above the horizontal meridian on the

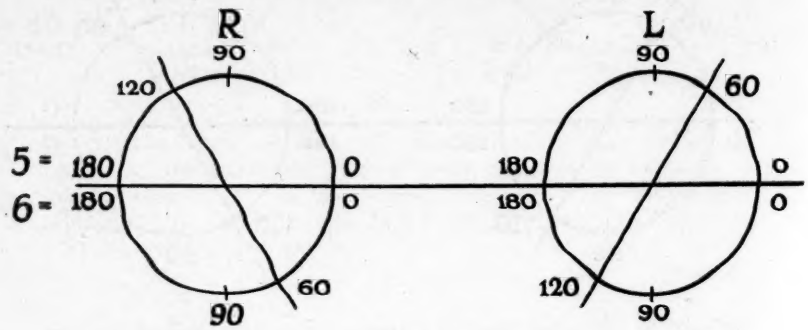

FIG. 7.

nasal side in the right eye and on the temporal side in the left eye, the scale being invariably marked from the point of view of the observer facing the patient. In order to use this method it is unnecessary to have trial frames with their graduated arcs above the horizontal, as shown in Fig. 7(5), which are very inconvenient

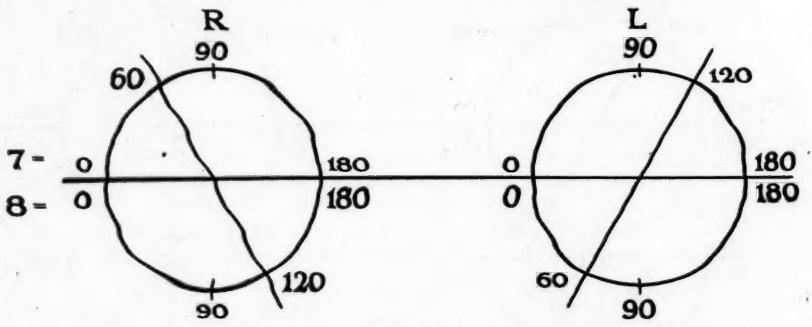

FIG. 8.

when changing the lenses. It is only required to have the usual trial frames graduated as below (Fig. 9), for this, as already pointed out, will give exactly the same numerical values for the angles as Fig. 7(5). With this standard notation, approved by the Optical Society in 1904, special prescription forms are unnecessary.

It will be noticed that the scale is the same for each eye. Such a method enables the optician to use a single machine for marking off the axes for the lenses of both eyes.

It is the standard notation of opticians in which all their machines are graduated, and it is into this notation that every prescription is translated by the optician before the order is passed on to his workshop.

All scientific optical and mathematical instruments are graduated in this notation.

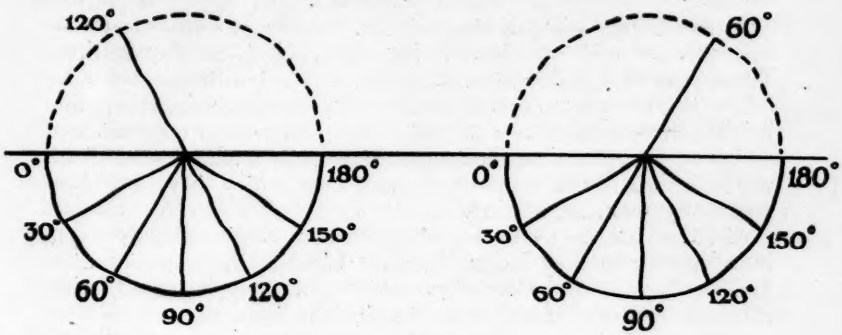

FIG. 9.

The Council, therefore, recommends the general use of this system of cylinder notation, namely:

That in which a similar notation is employed for each eye, the zero lying at the observer's left side and the scale being read below the horizontal with 90° at the bottom and 180° at the right side.

ANNOTATIONS

Lectures on State Ophthalmology

A series of lectures on State Ophthalmology was given in connection with the Royal London Ophthalmic Hospital School during the last fortnight in May. The first four were delivered at Moorfields and the last seven at the Royal Society of Medicine. The first part was concerned with the question of ophthalmology in relation to education, the opening lecture being given by Sir William Lister, who dealt with the subject from a general point of view, and set the keynote at a high pitch which was consistently maintained throughout the whole course.

Mr. Bishop Harman followed on May 18 and 19 with two

lectures on the education of children with defective eyesight, and these were full of interesting detail showing what a prodigious amount of laborious work has been put forward by himself, in conjunction with the public bodies with which he is associated, to enable such children to become useful members of the community.

Sir Arnold Lawson's lecture on May 20 illustrated similar efforts in connection with those blinded by accident, particularly during the war, with special reference to the work at St. Dunstan's, and enlightened his audience on the immense amount of care and forethought which is devoted to these cases.

At the Royal Society of Medicine Mr. Parsons gave three lectures, May 23, 24 and 25, introducing the subject of illumination. These were of a highly scientific order, which, if a little difficult to follow in some places, served as a salutary reminder that there are heights in ophthalmology to which many have never attained, but which at the same time should certainly create a stimulus in all of us to attempt the mastery of problems which have such an essentially practical bearing.

Mr. Leon Gaster (Secretary of Illuminating Engineering Society), in his lecture on May 26 on Practical Lighting in connection with Health, Safety and Efficiency, which was supplemented with excellent lantern slides, gave an insight into the numberless conditions where good illumination, based on scientific principles, plays such an important part.

Dr. Lister Llewellyn never fails to attract his audiences, and his lecture on Miners' Nystagmus on May 30 proved no exception to the rule; and he conclusively showed by slides, lamps, etc., how this disease is dependent on defective illumination.

Mr. Goulden on May 31 brought out by well authenticated statistics the enormous number of accidents to workmen from metallic foreign bodies, and how many of them could be avoided as well as an enormous sum saved to the State, by employing effective methods for the protection of the eyes.

Dr. T. M. Legge on June 1 gave us the benefit of his unique experience gained in the factories of the nation by demonstrating the inner workings of the Workman's Compensation Act in regard to Industrial Diseases with special reference to Glass Workers' Cataract.

Altogether this course of lectures was full of interest and instruction; and the School is deeply indebted to the lecturers for their kindness in consenting so willingly to come and help to widen the scope of its teaching; the only regret we have to record is that comparatively few took advantage of such an exceptional opportunity as these lectures afforded. We hope that if any similar course is given it may be made more widely known.

Ophthalmia Neonatorum

In a leading article dealing with the provisions which have been made for the early notification of ophthalmia neonatorum and urging the necessity of attending to the rules laid down, the *Lancet* says, inter alia: "Ophthalmic surgeons are agreed that with proper treatment of the eyes at birth the disease can be prevented, and cases of it should become exceedingly rare. There is general agreement that when it has occurred, given adequate and prompt treatment, damage to either eye should be very exceptional, and the the loss of an eye almost unknown. In these circumstances the occurrence of a case in any lying-in hospital should be made the subject of careful enquiry. Obstetricians and gynaecologists are not eye specialists, and it follows that every lying-in hospital should have a call on the services not only of a physician interested in paediatrics, but also of an ophthalmic surgeon, so that the nursing staff, the pupil midwives, and, last, but not least, any students attending the practice of the hospital, may have adequate instruction in the methods necessary to prevent the disease. If the first line of defence against ophthalmia is broken, efficient curative methods would thus be applied at the earliest possible moment."

With the foregoing reminder to lying-in institutions we very heartily agree.

We would further like to draw attention to the fact that the methods employed for getting immediate and adequate treatment for Ophthalmia Neonatorum are still very unsatisfactory. Cases occurring in practices of doctors and midwives are frequently not sent sufficiently soon to the special hospital provided in London by the Metropolitan Asylums Board, St. Margaret's Hospital, Leighton Road, Kentish Town, with the result that many cases are admitted when nothing can be done to save the sight.

It is to be hoped that the report of the Committee sitting on the "Prevention of Blindness" which is dealing with this subject inter alia, and is somewhat overdue, will make stringent recommendations to procure immediate treatment of the disease and also for the better education of students and midwives in its natural history, as was recommended in a resolution passed by the Ophthalmological Society in 1920.

ABSTRACTS

I.—FEBRILE HERPES OF THE CORNEA

Dœrr et Vöchting.—Studies regarding the virus of febrile herpes. (Etudes sur le virus de l'herpès fébrile.) *Revue Générale d'Ophtal.*, October, 1920.

Gruter, in 1913, reported the fact that herpetic keratitis of man could be inoculated on the cornea of the rabbit. Löwenstein later repeated Gruter's experiments, using various kinds of herpetic vesicles. He found that such inoculations in rabbits succeeded every time, whatever the form of febrile herpes, but did not succeed in herpes zoster. He also found that the different varieties of febrile herpes produce immunity in rabbits, and that the immunity is equal, in such a way that any kind of herpes immunizes against any of the other kinds. Experiments made with pemphigus, eczema, etc., were negative, and Löwenstein concluded that herpes febrilis is caused by a special virus belonging to the group of dermotropic excitants *(excitants dermotropes)*. Microscopically the results were uncertain.

Dœrr and **Vöchting** have further studied this question. Their experiments are of great interest, and may here be summarized.

Of six experiments to prove the transmissibility of herpes from man to rabbit, four were positive and two negative. The two failures are attributed to the transference being made too late in the evolution of the human herpes. The authors conclude that the evolution is very rapid. Experiments were then made from rabbit to rabbit, and these all succeeded provided the inoculation was made early, one or two days after the commencement. Further, an erosion of the cornea is not essential. If the transference is made into the conjunctival sac only, the incubation is longer, and, mainly, the resulting infection is of the conjunctiva. On the other hand, the attempt to transfer the herpes from the cornea of the rabbit to other parts, such as the lip and nostril, did not succeed. A very curious fact is that when the virus is injected into the anterior chamber or vitreous, the result is corneal herpes, not an intraocular infection. The result stated by Gins, that vaccinia injected into the blood stream can be produced in the cornea merely by scratching it with a sterile needle, cannot be reproduced for herpes. The authors conclude that the herpetic virus has the character of a parasite which attacks the epithelium, to a much higher degree than that of vaccinia. Dealing with regional immunity, which was asserted by Löwenstein to take place in rabbits, the authors have found, in spite of the fact that regional immunity

certainly does not occur in herpes febrilis of the human subject, either of the cornea or of the lip, that Löwenstein is right in his statement for rabbits. Further, herpetic infection of one eye alters the strength of the reaction of the other eye: the length of the incubation period is prolonged, and the herpes is frequently of such a benign form that one has to inoculate a healthy eye of another individual in order to make sure that the reaction has been positive.

The final paragraph of this article appears to the reviewer to be of very great interest: "The importance of regional immunity in the diagnosis of the herpetic virus, and in its differentiation from other viruses with identical action on the eye of the rabbit, does not require to be much enlarged upon. If one inoculates the cornea of a rabbit with a mixture of herpetic virus and vaccinia, the two infections are produced simultaneously. One can also produce herpetic keratitis on a cornea infected with vaccinia keratitis, and *vice versa*. It is easy to isolate the components of a mixture of virus by inoculating them upon corneae immunised against herpes or against vaccinia, and thus without difficulty to make a differential diagnosis."

ERNEST THOMSON.

II.—SQUINT, MANCINISM, AND TUBERCLE

Rivers, W. C.—Squint, mancinism, and tubercle. *Lancet*, February 12, 1921.

Rivers, in his recently published book, maintained that squint was about three times commoner in consumptives, and probably also in consumptives' parents, than in the non-tuberculous; that such squint seemed to favour the left eye; and that tuberculous squinters were left-handed, and gave a family history of left-handedness oftener than did non-squinters. Rivers, in the present article enlarges on this theme as the result of the examination of more extensive material. The article is choked with statistical statements in the text which make it extremely difficult to follow. One may be content to transcribe some of the statements and make some obvious comments on them.

"Not stopping then to do more than note the apparently new fact that squint is greatly commoner in children than in adults, we reach the first conclusion that in the subjects of some forms of tubercle squint is as much as twice or thrice as frequent as in normal persons."

It is, surely, not a new fact that squint is commoner in children

than in adults. The spontaneous or therapeutic "cure" of squint in children is one considerable cause of this.

"It seems fair then to conclude that squint is excessive in lung tubercle and in lymphatic gland tubercle, but not in bone and joint tubercle."

"Squint is excessive amongst the clinically non-tuberculous mothers (at any rate) of non-squinting consumptives."

"I have now particulars of 65 phthisical squinters; 48 of these squinted with the left eye. As to the normal lateral distribution of squint, the text-books appear to be silent . . . . . It would appear quite likely that left-eyed preference is a characteristic of squint generally, not only of squint in the tuberculous."

The reviewer has brought out this same predominance of left squints in school children in an article published in the BRITISH JOURNAL OF OPHTHALMOLOGY, July, 1919, and more completely in the *Annual Reports* of the Education Authority of the County of Lanark, 1914-15, and 1916-17 (Statistical Tables).

The author's statistics of the association of left eye squint with mancinism (left-handedness) are interesting, and he concludes regarding this that "whether in phthisical or in non-tuberculous subjects, left-eyed concomitant strabismus is somewhat associated with mancinism and with a family history of mancinism."

What then do we gather to be the author's point regarding squint and consumption? It seems to amount to this, that the development of clinical tuberculosis is favoured by an abnormal constitution, of which squint is a sign. Squint is "a mark of general tendency to abnormality." Mancinism has been reckoned among the stigmata of degeneracy. And we have the association of left-eye squint with mancinism.

The reviewer cannot help thinking that some of the author's arguments are unduly stressed. If he is going to associate concomitant squint with tubercle the association ought to be between tubercle and the optical conditions which most of us think have something to do with squint. Squint is unusual in emmetropes. Ought he not to begin at the beginning and seek to know why because, say, one cornea has a curvature somewhat different from the other, this, with the resulting visual confusion and the ultimate squint, should be a sign of abnormality; and why, if associated with left-handedness, a left-eye squint (that is, in effect, a left-eye optically inferior to the right) is to contribute to the signs of degeneracy? Should he not also seek to explain why eyes which exhibit extremely high hypermetropia, and which on that account might themselves with some show of reason be considered "abnormal," and possibly even "degenerate," so frequently do not squint?

Again, let us examine the following startling statement: "Take

only the significant phenomenon of squint being much commoner in ordinary school children than in ordinary adults. It might have been predicted; we know from school medical officers that very few working class children get cured of squint, but we also know that an undue proportion of these squinters are destined to find the asylum and the prison. What use does preventive medicine make of that knowledge? Now add that an undue proportion will also become tuberculous or the mothers of the tuberculous, and that into the bargain there is probably a considerably higher general mortality amongst them than amongst their school fellows. The neglect becomes serious." The author does not specifically refer us to authorities for these statements, unless they are contained somewhere in the 25 items of the bibliography.

Can we, ophthalmologists, believe that a child with a squint has, because of the squint itself, a better chance of going to prison, going to an asylum, or dying young, than a child who does not squint? One is not justified in disputing statistics, but only in questioning the interpretation of them. Is not the difference in the percentage of squinters between the juvenile and the adult population more rationally explained, as a whole, by spontaneous or therapeutic "cure" than by suggesting that it is largely due to so many of the juvenile squinters ending in the prison or the asylum? May it not also be the case that the people who reach these institutions are just the people whose parents neglect the eyesight of the children? With regard to tuberculosis, with regard to excessive mortality, what is the connection between these and the optical conditions which are associated with squint? If Dr. Rivers were to take 100 non-squinting children at random, and bandage their left eyes for a week or two (a procedure which cannot be advised) he would assuredly produce a percentage of left-eye squints. Would each of these squints be "a mark of a general tendency to abnormality," and would one be obliged to consider that the expectation of life of these children who squinted because of the bandaging had been reduced?

Further, does the school medical officer know anything about the future of individuals between the time of leaving school and full adult life? In any case, what is meant by the "cure" of squint? If a squinting child becomes a non-squinting adult—not such an uncommon occurrence after all—his optical defect, in all probability, has not been removed. His corneal curvature remains the same. Everything remains the same except the position of the eye. Yet, whether through the natural growth of his orbits or as a result of optical or other treatment, the individual would have passed out of the valley of the shadow, of death, tuberculosis, the prison, or the asylum. That is, of course a *reductio ad absurdum*.

The reviewer is persuaded that concomitant squint is largely a

preventable accident, preventable if we knew the optical condition of every young child. He is totally unable to see how ordinary concomitant squint or its causative factors, factors which, be it noted, do not come into action as a rule for several years after birth, can be regarded as a mark of a general tendency to abnormality, any more than refractive errors in general can be so regarded, and considers that the author should seek for some different interpretation of the statistics which he has collected.

ERNEST THOMSON.

III.—ON THE DEVELOPMENT OF THE FISSURAL REGIONS

Mann, I. C. (London).—On the development of the fissural and associated regions in the eye of the chick, with some observations on the mammal. *Jl. of Anatomy*, January-April, 1921. Vol. LV, p. 113.

This paper deals with the process of closure of the choroidal fissure as observed in chick, mouse, and human embryos.

In the chick, as is well known, the level of attachment of the optic stalk to the developing optic cup alters greatly during development, being at first at the inferior part, and later half way up the posterior surface of the globe. The formation of the lower retinal fields, therefore, occurs by extension of the margins of the cleft. This is not exactly equal on the two sides of the cleft, being slightly greater on the posterior side, since the direction of the fissure alters from a right angle, in early embryos, to 70° with the horizontal downwards and forwards, in the adult hen. The fissure closes first in its intermediate portion, leaving a proximal opening for the choroidal vessels, which pass out again at the distal opening of the fissure. This later closes; the vessels are cut off here and become shorter.

Owing apparently to more rapid growth of the inner layer of the optic cup, this layer becomes everted in the upper part of the fissure, so that on the outer surface of the eye a non-pigmented area is visible on either side of the cleft, continuous with the true outer, pigmented layer some little distance from the cleft margins. When closure of the cleft occurs fusion therefore takes place between non-pigmented areas only.

Comparatively large areas of the non-pigmented layer fuse so that the line of the fissure is marked by the heaping up of this layer, which forms two ridges, one (the *crista intraocularis*)

projecting into the vitreous, the other on the outside of the globe. In the region above the entering vessels the developing nerve fibres grow into this external projection (since it is in origin directly continuous with the inner fibre-bearing layer of the optic cup) and run up on the outside of the eye to reach the attachment of the optic stalk. These fibres form the *cauda* of the adult nerve. The fibres of the developing *cauda* do not reach the cleft by following the inner surface of the retina, but pass deeply into its substance, thus cutting off the cells of the *crista intraocularis* from the rest of the inner layer. This crest grows into the vitreous, becomes vascularised from below upwards by the proximal ends of the choroidal vessels, develops pigment and becomes folded upon itself, forming the pecten, which is thus seen to be primarily ectodermal in origin. In the adult hen the " roots " of the pecten can be seen to be continuous with the neuroglial septa of the optic nerve.

Below the entering vessels the eversion of the inner layer is not so great, no nerve fibres grow into it and no *cauda* is formed. The internal ridge also disappears, no sign of the line of the fissure remaining.

In mammals also the eversion of the inner layer in the lips of the fissure occurs. It is very well seen in the mouse. In 13 mm., 15 mm. and 16 mm. human embryos it is present as a small mass of non-pigmented cells continuous with the pigmented layer and situated immediately below the nerve. In man, however, no nerve fibres normally grow into this everted area and so no *cauda* is formed to the optic nerve.

IV.—GUMMATOUS ULCERATION OF THE TARSUS

Maucione, Dr. L. (Naples)—On gummatous ulceration of the tarsus. (Contributo allo studio delle alterazione gommose ulcerate del tarso.) *Arch. di Ottal.*, July to September, 1920.

Maucione has had the opportunity of seeing three cases of gummatous ulceration affecting the tarsus. He points out that the disease is of considerable rarity, as he has only been able to find twenty cases recorded, and, in addition, that the differential diagnosis may present a good deal of difficulty.

The first case was that of a man of 27, who had acquired syphilis seven years before the tarsal affection had developed. This was treated with mercurial injections and " J.K." Seven years later he suffered from severe bone pains and calomel injections were repeated without benefit. The " Pollini " treatment was then tried, but after the sixth dose the right upper eyelid became swollen and some days later an ulcer was noticed on the corresponding part of

the tarsal conjunctiva. This got well under treatment, but relapsed a few months later, and this time remained unaffected by mercurial treatment.

When first seen by Maucione there was a swelling in the right upper lid about the size of a coffee bean. On everting the lid, which could be done easily and with little pain, there was seen a circular ulcer of about 7 mm. in diameter with sharply defined undermined edge and raised above the surrounding conjunctiva, which was a little hyperaemic. The surface of the ulcer was covered with a whitish adherent exudation, the removal of which caused a slight flow of blood-stained lymph. The Wassermann reaction was strongly positive and the skin reaction to tuberculin was negative. Microscopic examination of the exudation and ulcer fragments gave no positive results. The injection of 60 ctg. of neosalvarsan was followed by a complete cure in ten days. Twenty-two days later a relapse occurred in which all the features already described were reproduced. This was treated by two further injections of neosalvarsan in doses of 45 and 60 ctg. A fortnight later the ulcer had disappeared and the patient has remained well since.

The second case was that of a man of 24 who had contracted syphilis four years previously. This patient's father had also suffered from syphilis, and of his ten children seven had died in infancy. The treatment adopted in this case was the same as in the first case. Four years after the primary lesion the right superior eyelid became swollen. This subsided without treatment, but a recurrence took place two months later, this time in the right inferior lid, with rapidly increasing ulceration near the free margin. Maucione then saw him and gives a description of the appearance which was practically identical with that observed in the first case. Wassermann reaction positive, tuberculin skin reaction negative. Microscopic examination of scrapings showed nothing of importance. A complete cure followed a single injection of neosalvarsan.

The third case was that of a man of 23. In early youth he had suffered from periostitis of the leg which required scraping and a protracted course of "iodo-iodurate" injections. He contracted syphilis a year before the tarsal affection appeared. When this appeared he was under an intensive mercurial treatment for skin symptoms. When seen by Maucione there was an ulcer in the middle third of the free margin of the right upper lid. When the whitish surface exudate had been removed a cavity appeared which bled readily and extended about 4 mm. within the tarsus involving both the skin and conjunctiva of the free margin of the lid. Wassermann reaction was negative, probably on account of the recent intensive mercurial course, skin reaction to tuberculin was negative. As in the previous cases microscopic examination was of no assistance. Two doses of 45 and 60 ctg. respectively of

neosalvarsan not only caused the ulcer to heal but also cleared up the skin lesions which had not yielded to the previous intensive mercurial treatment.

The paper concludes with some comments on the differential diagnosis, the nature of the lesion, and the value of arsenobenzol preparations in the treatment after the failure of mercurial injections.

Photographs of the first two cases and a bibliography accompany the paper.

E. E. H.

V.—PRIMARY EPIBULBAR CARCINOMA

Calhoun, Phinizy (Atlanta, Ga.).—Primary epibulbar carcinoma. *Amer. Jl. of Ophthal.*, February, 1921, p. 101.

Calhoun reports a case of epibulbar carcinoma in a man, aet. 64, which occurred as a rapidly increasing tumour of the lower lid, accompanied by defective vision. Eighteen years before author's examination, the patient had undergone operation for a bulbar growth which had developed at the site of an excised pterygium. This growth had been diagnosed malignant, but there was no recurrence until six weeks before examination by the writer. On examination, the lid tumour was found to be of the size of a hazel nut and firmly adherent to the globe, and encroaching slightly upon the limbus at its lower part. There were old trachomatous scars in the upper lid, and pannus was present. Patient refused enucleation, and the tumour was shelled out. Following confirmation of diagnosis of malignancy by pathologist's report, radium was applied two days later at the site of operation (20 mgm. for 1½ hr. to raw surface). Seen two months later, cornea had cleared, and vision improved. Fundus was normal.

A year later patient sought advice on account of an enlarged gland in the preauricular region. Pannus had returned and a small growth could be made out at the site of the original tumour (the lid being adherent to the globe at this point).

Patient again refused enucleation, and the tumour (with the gland) was removed under local anaesthesia.

No recurrence of any kind had taken place when the patient was seen six years after the first operation.

Several photo-micrographs of the tumour and gland tissue are given.

Calhoun, being in some doubt as to the correctness of his diagnosis of malignancy, submitted sections of the tumour and

gland to several pathologists who completely confirmed his opinion.

He alludes to the controversy that obtains in regard to such tumours, some surgeons holding that penetration of the globe rarely takes place, and others—among whom he quotes Parsons and de Schweinitz—considering that peneration is frequent.

Calhoun accepts the teaching that if the tumour be small and at the limbus, a deep excision may be made and developments carefully watched, but that if the growth be at a distance from the limbus, even if of larger size, it may be excised with confidence in the result.

[Radium treatment in the present case may have played a considerable part in the cure of the condition, even accepting the diagnosis of malignancy as established.]

J. HAMILTON MCILROY.

VI.—CONGENITAL WORD BLINDNESS

Macleod, R. A., M.D. (Gympie, Queensland).—Congenital word blindness. *Med. Jl. of Australia*, June 26, 1920.

Macleod gives notes of three cases of congenital word blindness observed by him in the same family. He points out the difficulty of training such patients to earn their living, and that, in consequence of their failure to do so, the burden falls on the public purse. While two of his patients were mentally deficient in other respects, one of them was said to have possessed average intelligence and an exceptionally good memory. So good was his memory that his defect was not at first discovered, as he appeared to read from memory of what the teacher had said. As a result of being taken away from school at ten, he had, four years later, degenerated and appeared weak-minded. All three were sent for six months to a lunatic asylum as they were difficult to manage at home, but no benefit followed. There was no hereditary history, and the other five children were all normal. Macleod gives an account of Gaskell's theories of nervous reflexes, and suggests that the development of fresh collaterals by education might have possibilities in these cases. He was also much struck by an account of Pavlov's experiments on the acquirement of cerebral reflexes by the lower animals, and considers that the same principles might be used in the education of the human defective. "The patient should be kept in a room by himself and the reflex between the word and the utterance of a sound slowly brought about without the presence of any advent'tious noise, words of encourage-

ment, evidence of impatience or unnecessary stimuli falling on the eye or ear. Every avenue of approach to the cortical cells may and should be brought into requisition, as the larger the number of associations the more easily, and perhaps at the same time the more vividly, would the impression be recalled."

E. E. H.

VII.—HUMAN MYIASIS.

Wright, R. E., and Patton, W. S.—A case of myiasis of the frontal and ethmoidal sinuses and the orbit, with an entomological note. *Ind. Med. Gaz.*, February, 1921.

Wright's patient was a Hindu woman of 30, whose supra-orbital and inter-orbital regions were alive with maggots; the bones had been extensively attacked; the surrounding sinuses were freely involved; the dura was not exposed. The eyeballs were exposed, proptosed, and pushed outwards; the cornea of one of them was ulcerated. There was marked oedema and a foul discharge. The cavity was irrigated with permanganate solution, and turpentine applications were made; 50 maggots were removed the first day and 30 the next. Chlorine solution was substituted for the permanganate and iodoform packing adopted. The prognosis is stated to be good for life, but poor for sight. The extrinsic muscles of the eyes had been extensively destroyed. **Patton** identified the maggots as the mature larvae of *Chrysomyia bezziana* Villeneuve, the specific-myiasis producing Calliphorine of India, which deposits its eggs only in the diseased tissues of man and animals. He states that it is clear that the eggs were laid directly on an ulcerated surface, resulting from the lancing of an abscess on the patient's eyebrow, and not within the nose. At the same time a nasal discharge, such as the patient had, serves to attract the female *bezziana* to oviposit inside the nostrils, the larvae then penetrating into the sinuses connected with the nose. He asserts that these larvae do not penetrate bone or cartilage, and that the destruction of these hard tissues, as seen in this case, is brought about by dissolution through the action of bacterial ferments. All muscid larvae confine themselves to fluid food, the substances on which they feed being dissolved by salivary and bacterial ferments. The patient had noticed the larvae for three days, and the eggs must have been laid ten days earlier.

R. H. ELLIOT.

VIII.—HEREDITARY POSTERIOR POLAR CATARACT

Ziegler, S. Lewis, and Griscom, J. Milton (Philadelphia).—Hereditary posterior polar cataract, with report of a pedigree. *Trans. Amer. Ophthal. Soc.*, Vol. XIV, Part i, 1915, p. 356.

(7) *Apropos* of the explanation of hereditary cataract, **Ziegler** and **Griscom** point out there are two schools: the one believing that the cause is toxic, and the other believing that it represents an arrest in development of the lens, due to some inherent abnormality in the germ-cell. There is strong evidence in support of both theories. But it is probable that neither one singly explains every case of congenital cataract, while in some instances both may be active factors.

In the cases reported the opacity was situated on the posterior capsule of the lens, and varied from a small round dot at the posterior pole to a dense circular disc covering the central third of the posterior capsule.

The original member of this family came to the United States from France about 1810, but nothing is known concerning the condition of his eyes. The first member of the family known to have congenital cataracts was a son of this original settler. In the second generation there were nine members, of whom six were affected. The third generation contained thirty-one members, of whom ten were affected. The fourth generation contained twenty-three members, of whom seven were affected. A study of the pedigree showed that there were sixty-four members in the four generations, of whom twenty-four (37 per cent.) had congenital cataracts. Of those affected, 43 per cent. were females and 34 per cent. males. The rule of "once free always free" was followed, since in no instance did normal parents produce affected children.

Charles B. Davenport reported concerning the pedigree that the cataract in this family behaved as dominant. So long as these people with early cataract married, they were bound to perpetuate their trait in half their descendants. Normals, on the other hand, need have no fear of marrying and having children.

S. S.

BOOK NOTICES

Squint: its causes, pathology and treatment. By CLAUD WORTH, F.R.C.S., Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields. Fifth Edition. Baillière, Tindall and Cox. Price 12/6.

We welcome the fifth edition of Worth's book on Squint. In

comparing it with the first edition issued in 1903, we note that there are now 242 pages instead of 229. In the appendix appear the results of 100 consecutive advancement operations.

The chapter on operations has been much enlarged. In the first edition the author advised tenotomy to be combined with advancement in severe cases to prevent enophthalmos, now he hardly ever performs tenotomy at all, but where a large effect is necessary in convergent strabismus he advances both external recti, the second operation following the first at an interval of 10 days. After an advancement he keeps both eyes covered for 10 days, and if at the end of this time a further operation is necessary, both will be covered for 20 days. This is a rather serious matter. The author still performs his advancement operation as at first published, but now uses special straight cutting needles for the insertion of the scleral sutures.

The book is well printed and free from errors. The illustrations are the same as those in the first edition.

E. W. B.

Bulletins et Mémoires de la Société française d'Ophtalmologie. Thirty-third year, 1920. Paris: MASSON ET CIE., Editeurs, Libraires de l'Académie de Médicine, 120, Boulevard Saint Germain, 6e.

The *Bulletins* of the French Society of Ophthalmology for 1920 have now been published. The handsome binding in which the volume was issued before the war has been dropped, probably on the ground of expense, and a paper cover is substituted. One of the first things to do, therefore, is to have the volume properly bound. Only six ophthalmic surgeons in Great Britain appear in the list of members, which is a pity, as the French Transactions always contain some valuable articles, and admission to the Society is quite easy. The present number contains 255 pages, of which almost half are occupied by the excellent *rapport* by Dr. Gonin, of Lausanne, on Idiopathic Detachment of the Retina. The rest of the contents consist of articles of varied interest, including one by Dr. Barraquer, of Barcelona, on the operation which he has called phakoerisis. Extracts from the more interesting papers will be published in this Journal from time to time.

CHARLES KILLICK.

The Medical Annual, 1921. Bristol: John Wright & Sons, Ltd. London: Simpkin, Marshall, Hamilton, Kent and Co., Ltd. Price 20/- net.

This useful work of reference has again made its annual appearance and maintains the high standard set in its previous thirty-eight years of publication. The ophthalmic section has this year been undertaken by Burdon Cooper. The most important article

deals with the medical treatment of cataract. The chemical pathology of cataract must necessarily form the basis of any possible medical treatment of this affection. Burdon Cooper has himself done some research work on the subject, and in this all too short summary gives an account of the work of other writers, together with a brief note of Burge's excellent paper on the injurious effect of ultra-violet light on living tissue (*Bull. Nela Research Lab.* 1919, Vol. I., paper 61). Other articles deal with diseases of the conjunctiva, glaucoma, diseases of the retina and general therapeutics. In addition to several text illustrations, there is an excellent coloured plate of congenital multilocular cysts of the retina reproduced from the *Journal of the American Medical Association.*

The only criticism we feel inclined to offer on the ophthalmic section is that it is rather more suited for the specialist than for the general practitioner.

E. E. H.

NOTES

North of England Ophthalmological Society

THE following gentlemen were elected to the Council of the Society at the annual meeting held in April. Session 1921-1922:—President: J. Gray Clegg, Manchester; Vice-President: Geo. H. Oliver, Bradford; Hon. Treasurer: Harry Lee, Leeds; Hon. Secretary: Percival J. Hay, Sheffield; Council: A. E. Burroughs, Liverpool, and H. Caiger, Sheffield.

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Appointment

A. CHRISTIE REID, D.O. (Oxon.) has been appointed surgeon to the Nottingham Eye Infirmary vice Col. H. Herbert (resigned).

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Corrigendum

IN the account given in the last number of the Journal of the meeting of the Ophthalmological Society it was stated that certain slides of malarial parasites in the intraocular vessels were shown by Professor Shattock, whereas the specimens were obtained by Professor Leonard S. Dudgeon at Salonika and exhibited by that gentleman.

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Dr. Freeland Fergus

THE Senate of the University of Glasgow has resolved to confer the honorary degree of Doctor of Laws upon Dr. Freeland Fergus, President of the Royal Faculty of Physicians and Surgeons of Glasgow.